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Volume XII. NOVEMBER, 1928 No. 11

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ANNUAL SUBSCRIPTION $2 SINGLE COPIES 25 CENTS

Entered at the Postoffice at Phoenix, Arizona, as second class matter.
"Acceptance for mailing at special rate of postage provided for in section 1103, Act of October 3, 1917, authorized March 1, 1921."

CLASSIFICATION AND DIAGNOSIS OF FEEBLEMINDEDNESS

J. G. WILSON, Surgeon,
U. S. Public Health Service
El Paso, Texas.

(Read before The El Paso County Medical Society, at El Paso, Texas, March 19, 1928, and before the Texas State Medical Association, at Galveston, Texas, May 8-10, 1928. Published, also, in the Texas State Journal of Medicine, for November, 1928.)

By feeblemindedness we mean "a state of restricted potentiality for or arrest of cerebral development in consequence of which the person affected is incapable at maturity of so adapting himself to his environment or to the requirements of the community as to maintain existence independently of supervision or external support."

This is the sociological definition of feeblemindedness. It is quoted from Tredgold and is identical with that contained in the English Mental Deficiency Act of 1913. In the United States we have no Federal Act defining feeblemindedness and the various state laws recognize the condition without setting it forth in scientific terms. However, the American Association for the Study and Prevention of Feeblemindedness, The National Committee for Mental Hygiene, and such eminent individual American authorities as Healy, Goddard and Terman, all agree that the definition should in fact contain the main points as set forth in the English law.

It will be noted that this definition embraces two main points:

(1) The mental defect must be due to some interference in the natural development of the brain, thus not only clearly differentiating it from dementia and the insanities but also making it imperative that the cause exist from birth, infancy or early childhood.

(2) The defect must be of sufficient gravity to prevent the individual assuming his proper place in society without the care and supervision of others.

Although there is no sharp clean-cut dividing line between the higher grades of feeblemindedness and the lower degrees of normal mental ability, there is a general consensus of opinion that the differences are sufficient to warrant the division of all persons into eight main classes according to intellectual ability. That is, we are, all of us, whether sane or insane, either

1. Idiots
2. Imbeciles
3. Morons

or

4. Borderline cases
5. Dullards
6. Normals
7. Supernormals
8. Geniuses.

The term, Feeblemindedness, then, is a generic term embracing three grades or degrees of inherent mental defect, to-wit: idiots, imbeciles and morons.

Sociologically considered, and speaking in general terms only, an idiot is a feebleminded person whose defect is so great that he is unable to guard himself against the common physical dangers of life. From the same standpoint and in the same general terms, an imbecile is one who is able to guard himself against these dangers but requires aid or constant supervision in doing the simplest tasks, while a moron can do routine tasks without supervision but is unable to plan or meet unexpected new situations without aid and supervision.

But these sociological or industrial definitions do not go far enough. Many feebleminded persons living in crude or primitive rural environments are able to pass as normal among their own fellows, but, when removed to the more complex milieu of our average cities or towns and compelled to compete with persons of higher intellectual attainments, their inherent deficiency becomes evident.

Therefore, in order to have a clearer idea of just what constitutes an idiot, imbecile, or moron, some purely psychological measuring stick is necessary. We have such an instrument in the standardized performance

tests of Binet-Simon. These have been modified by Terman of Leland Stanford University and it is this modification which is regarded by the majority of American workers as the most trustworthy for the examination of our own native born.

From an application of these tests we determine the subject's so-called "mental age" or performance ability", and his "intelligence quotient." Measured in this way, an adult idiot's mental age or performance ability ranges from that of a normal newborn to a normal three-year-old, while an imbecile's ranges between three and seven years and a moron's between seven and eleven.

The intelligence quotient, familiarly spoken of as the "I. Q." is the figure obtained by dividing the mental age by the actual or chronological age. According to this standard idiots have an intelligence quotient ranging from 0 to 20; imbeciles, 20 to 50; morons, 50 to 70; borderline cases, 70 to 80; dullards, 80 to 90; normals, 90 to 110.

The following table summarizes what I have just said about the various grades of mental defect and at the same time indicates their etiology and chief clinical varieties.

TABLE 1

CLASSIFICATION OF THE FEEBLEMINDED ACCORDING TO

I. MENTALITY	ETIOLOGY	III *CLINICAL VARIETY*
1. *Morons* (I. Q. 50-70) Performance Ability 7-11 Years. Can do routine tasks without supervision, but can not plan.	1. Primary or inherent (90%) (Characterized by pathological germinal variations: Due to defective heredity).	1. Simple 2. Microcephalic 3. Mongolian 4. Epileptic
2. *Imbeciles* (I. Q. 20-50) Performance ability 3-7 years. Require supervision and aid in doing the simplest tasks but can guard themselves against ordinary dangers.	2. Secondary or Accidental (10%) (Characterized by gross cerebral lesions or defective cerebral nutrition: Due to toxic or mechanical causes, specific glandular defects, or deficient sensory stimulation).	5. Amaurotic 6. Syphilitic 7. Hydrocephalic 8. Paralytic 9. Cretinoid 10. Nutritional 11. Isolated
3. *Idiots* (I. Q. below 20) Performance ability under 3 years. Unable to distinguish and avoid ordinary physical dangers.		

II

The next table, prepared by the National Committee for Mental Hygiene, elaborates the etiological factors mentioned in Table 1.

TABLE 2

CAUSES OF MENTAL DEFICIENCY

Heredity—
- Mental defectiveness
- Insanity
- Epilepsy
- Alcoholism
- Syphilis

Congenital Defects—
- Hydrocephalus
- Miirocephalus
- Defects in cerebral substance
- Defects resulting in mental deprivation
  - Blindness
  - Mutism
  - Deafness

Injuries During Birth—
- Fracture of skull
- Compression of brain
- Cerebral hemorrhage
- Asphyxia

Diseases during Infancy—
- Acute Infectious diseases:
  - Scarlet fever
  - Pneumonia, etc.
- Diseases directly affecting the brain:
  - Infantile cephalitis
  - Meningitis
  - Syphiis
  - Infantile convulsions, etc.
- Epilepsy
- Malnutrition

Thyroid Insuficiency

Injuries during Infancy

There is no abnormal physical condition in which an exact diagnosis is more important than in feeblemindedness, for upon this

feature hangs, (1) the possibility of cure of the individual himself; (2) the prevention of much crime and economic waste; (3) the moral responsibility of many delinquents; (4) the prevention of the propagation of feebleminded stock.

By reference to Table II, one may infer that cure is not entirely out of the question in a certain small proportion of cases—notably, those dependent upon deprivation, such as congenital blindness and deafness, and to a lesser degree those caused by syphilis, birth injuries and thyroid deficiency—and that proper training will greatly relieve the situation in all cases except the lowest grades of idiocy.

But aside from cure or relief of the condition itself, the greatest importance attaches to the prevention of crime and economic waste.

These persons should be classified and properly disposed of early in life, long before they have wasted the tax-payers' money and the teachers' efforts in a vain attempt to give them an education which they are incapable of receiving, and long before they have had the opportunity to become juvenile delinquents.

While the importance of proper diagnosis is evident, it is not so easy to make as might at first appear. Mental deficiency must be differentiated from the lowest grades of normal development and from the borderline cases. It must also be differentiated from certain eccentricities and transient personality defects of adolescence, and those forms of dementia which occur in epilepsy, juvenile paresis, and dementia praecox. Then, too, the social mal-adaptation or failure to progress in school may not be due to inherent mental deficiency at all, but solely to lack of interest, poor instruction or some removable physical defect. All these possibilities must be held in mind and eliminated before forming an opinion.

The character defects and intellectual status of the subject's ancestors and collateral relatives should be carefully considered and balanced with his own sociological and industrial history. After this is done, his "mental age" and "intelligent quotient" should be obtained and all the evidence weighed together, not unduly stressing one factor to the exclusion of others.

Some weight should, of course, be given to the general appearance, but in doing this it should be remembered that normals frequently look idiotic and that the feebleminded often look normal.

The coincidence of paralytic conditions and mental defects does not necessarily mean that the latter is dependent upon the former. The same causes, nutritional or congenital, which were responsible for an imperfectly developed cerebral cortex, may result in an inadequate musculature or malformed osseous system. The gait of the lower grades of the feebleminded is notoriously slouchy and incordinated and club feet and paralyses of various degrees often exist without gross cerebral or spinal lesions. Cerebral decomposition and orthopedic operations on the feebleminded should therefore be undertaken in only very carefully studied and selected cases and the orthopedist should not lay too great stress on the purely objective physical deformities, for they may simply fall into the general classification of "stigmata"—incapable of correction and of diagnostic significance only.

In regard to stigmata, their presence may or may not have diagnostic import. One must be very careful not to unduly emphasize the significance of supernumerary fingers, simian heads, or abnormal ears.

This young man, shown in Fig. 1, reported by Knox in The New York Medical Journal, June 14, 1913, was suspected of feeblemindedness because of his general appearence as he came along the line at Ellis Island, and was turned aside for a complete mental examination (note the shape of the

head and the malformed helix). Yet a competent mental examination showed him to be of normal mental balance, above the adult average for his race, and able to speak three languages fluently.

However, when many different stigmata occur in the same person they usually have marked significance. A cleft palate alone means nothing; but a cleft palate, a clubbed foot, supernumerary digits and small receding forehead, in the same individual, would very likely betoken a deficient mental as well as physical development. No mental tests are necessary to create the belief that the man shown in Fig. 2, described by Tredgold in his work on Mental Deficiency, is sadly lacking in cerebral ability. Here we have a combination of stigmata which would arouse anyone's suspicion. He is microcephalic, has distorted ill-shapen teeth, asymmetrically placed orbits, a supernumerary thumb and limply hanging lower jaw. His whole attitude betokens a mental defect and we are not surprised to learn that he was in fact an imbecile who was unable to talk and required constant care and supervision.

But even a silly attitude accompanied by stigmata such as this man displayed, must not be interpreted alone.

The girl shown in Fig. 3 certainly looks silly enough, with her small pig-like eyes, asymmetrical face, and thick wrinkled skin over the forehead. Any medical inspector of immigrants would be justified in turning her aside for a mental examination, and yet she was just a normal Slavic peasant girl who had not been in the United States long enough to learn the art of disguising an ugly face by framing it with marcelled hair, stenciled eyebrows and carmine lipstick.

Children convalescent from acute illnesses are frequently so dull and stupid that an inherent mental defect is simulated.

I think any one would have said that the appearance of a child admitted to my service at the contagious disease hospital at Ellis Island indicated a low grade of mental defect. She was suffering from a severe whooping-cough, followed by suppurative otitis media—upon which an unusual train of mental symptoms developed, characterized by great apathy, stupidity and katatonic posturing. But as her physical condition improved her mentality also became normal and she was eventually discharged as sound in both mind and body. The history of the case was published in great detail in the Journal of Abnormal Psychology, July, 1926, and is another illustration of the necessity for careful study and prolonged observation before making a diagnosis of feeblemindedness.

In forming our conclusions about the feeblemindedness of a given individual, we are greatly helped by a complete family history. Ninety per cent of these cases fall in the hereditary group and, if we could always obtain a genealogical tree with biographical data extending back four or five generations, our task would often be greatly simplified. The influence of heredity is graphically shown in Fig. 4, which summarizes Goddard's Study of the Kallikak family. Here we have four hundred and ninety-six direct descendants from a normal father and mother with no feeblemindedness in any of them. These are contrasted with four hundred and eighty descendants from the same normal father and his feebleminded paramour, of whom one hundred

and forty-three were classified by Goddard as distinctly feebleminded. The last in the line was Deborah Kallikak—a high grade moron whose picture is shown here and whom I had the privilege of seeing personally when she was an inmate at the Vineland Training School fifteen years ago. You can see from her picture that she was a pretty, attractive-looking girl, with nothing in her general appearance to indicate low mental ability.

And this brings me naturally to the last and most important point I wish to make, and that is the actual value of the performance test which I have mentioned.

Judged by the Binet-Simon tests, Deborah Kallikak was a high-grade moron; judged by the manner in which she performed her tasks at the Vineland Institute she was also a high-grade moron. Her genealogy, with its long line of social misfits, also seemed to substantiate this diagnosis. But, in my opinion, the use of performance tests alone would not have been sufficient to fasten the moron tag upon her. Performance tests, especially the Stanford Revision of the Binet-Simon tests, when used by persons who know how to interpret them, are of enormous value. I would compare them to the stethoscope in making a diagnosis of tuberculosis. There are some cases of pulmonary tuberculosis which competent men correctly diagnose on auscultatory evidence alone, but they are not the doubtful cases. There are some cases of feeblemindedness which competent men can undoubtedly correctly diagnose on the evidence of performance tests alone, but they are not the borderline cases. And above all one must be careful not to draw hasty conclusions from inability to perform one or two so-called key problems.

One of the patients whom I am going to show you tonight not only looks the part, but she failed to arrange the pieces in Knox's imbecile form board test—a thing any normal five-year-old child should do. However, when put through the whole series of Stanford-Binet-Simon tests she showed an intelligent quotient of seventy-two and one-half and her real troubles seem to me to be concerned with her personality and environment almost as much as her mental make-up. Accurate diagnosis of her case is difficult but she is probably a borderline case.

In making a diagnosis, one must also be careful not to let his opinions be swayed because the subject is ignorant about public matters or news which he himself may consider vital.

During routine mental examinations of immigrants, I recently asked a series of forty young adult Mexicans, who was the president of their country. Thirty-one knew, but nine did not know, and yet these nine were mentally normal, as evidenced by their ability to perform the tasks involving foresight and planning. They simply did not care who the President of Mexico was, and that was the whole explanation.

So, in conclusion, I would say that performance tests have great value. Badly informed persons may ridicule them, just as Laennec's stethoscope was ignored and ridiculed when first introduced, but these tests have come to stay just as surely as did the stethoscope, and it is just as much the duty of the mental examiner to learn how to use and interpret them as it is the duty of the chest specialist to perfect himself in the use of the stethoscope.

Patients of the following types were exhibited.

1. Primary. Idiot (high grade); due to glandular deficiency.
2. Secondary. Imbecile (medium grade); paralytic type.
3. Primary. Moron (low grades); simple types due to hereditary defects.
4. Borderline cases.

CASES

IDIOT. This little boy is five years old. Unfortunately I know practically nothing of his family history. The records of the Salvation Army Home, where he lives, show that his mother is a Mexican woman, and that her husband is an American soldier who has deserted her. There are several other children older than he and perhaps one or two younger, although on that point I am uncertain. All the other children are said to be normal. The diagnosis of the *degree* of mental defect in this case is easy. He is an idiot. He cannot feed himself. He cannot make his wants known. He wets his bed and clothing the same as an infant. He utters no intelligible sounds. speech is not even attempted. However, the primitive feelings of hunger and thirst are present, and he shows some signs that indicate the possibility of eventually training him so that he can indicate his desires to go to the toilet. His idiocy is, therefore, not complete, for a *complete* idiot is nothing but a lump of flesh, lacking not only in all the fundamental organic instincts but also without any powers of attention and with complete incapacity for being taught.

The etiology of this case is especially interesting. At first glance one might think he was a cretinoid, but careful examination shows that he does not fall in that class. Although he undoubtedly represents some type of glandular deficiency, he lacks the typical cretinoid features. His skin is fine and soft to the touch: his nose is not broad and flat; his orbits, instead of being wide apart, are rather close together; his tongue is not fissured. What one is most impressed with is his fatness. His body could have served as a model for one of Michael Angelo's cherubs. Note the rounded sloping shoulders, the feminine hips, the marked amount of adipose tissue around the mammary glands, and the soft rounded contour of arms and legs. This is a case in which one is justified in suspecting deficient secretion from the pituitary gland. He certainly presents the opposite

features from acromegaly, and if, as he approaches puberty, the genitals fail to develop, his stature remains dwarf-like and he shows an abnormal sugar tolerance, our diagnosis will be confirmed, and we can definitely label him as mental defect accompanied by the syndrome of *Frohlich*, or *dystrophia adiposo-genitalis*. In the meantime, an x-ray might show some abnormality in the size of the sella turcica, and the administration of pituitary extract might possibly have some beneficial action on his condition. He will be transferred to the Texas State Institution for the Feebleminded within a few days and undoubtedly a more complete study will be made of him there.

IMBECILE. This girl is thirteen years old. She is suffering from a spastic paralysis of the lower limbs which, at first thought, one might suppose to be dependent upon a birth palsy. However there is no history of difficult labor and the positive evidence for primary defective germ plasm is so great that I believe that both her paralysis and mental defect have a common cause in a defective development of the centrol nervous system. She has a brother who does not progress at school as rapidly as he should, whose intelligence quotient is only 76.4, and who needs further study before he is labelled "normal." The mother is a credulous, ignorant woman, who impresses me as being a moron, although I made no formal examination of her and am not able to say positively that such is the case. However, the social history of the family indicates inability to adjust its members to adverse conditions. Domestic troubles and economic difficulties are so great and constant as to suggest the possibility of inherent mental defect on the mother's part as the primary cause of all their troubles. This thirteen-year-old daughter is an *imbecile*. In spite of her smiling face and winning ways, she is totally incapable of performing the simplest tasks without supervision or aid. At the time of my examination she could not count to ten without making a mistake. Here are some of her answers to questions:

2 plus 2 equals—3.

3 plus 1 equals—2.

3 minus 1 equals—4.

This problem was illustroted concretely by asking her if she had two apples and some one gave her two more, how many she would then have, and so on through the list, but the result was the same. She had no idea whatever of numbers. Her power of attention was of a very low order and her ability to discriminate between different kinds of diagrams practically nil. A diamond, a square, and a parallelogram all looked alike to her, and when asked to copy these figures from memory after they had been exposed to her, she made a rude shaped square for all of them. She cannot read or write her own name. Her speech is also defective, and, according to her mother, she made no effort to talk until she was five years old. When I examined her five months ago, I estimated her mental age as about five years and stated that I did not believe it was worth while to send her to school any more. I have not seen her since then until today, but I see no reason to change my opinion.

MORON. This little woman is only thirteen years and seven months old. I want you to take a good look at her and her four-months-old baby. The baby is, to all *external* appearances, perfectly normal, and so, I think, is the mother. She is a bright attractive *looking* person. She went through the six year old tests of the Stanford revision of the Binet-Simon tests like a breeze, answering every one of them—and then she stopped—stopped suddenly and completely. She failed on every one of the seven-year-old tests. Graded by her performance ability she is only six years old and her intelligence quotient is forty-six. She is, therefore, from this standard a high-grade imbecile, but her ability to perform routine tasks, and to feed and look after her own baby (under supervision), together with the fact that her intelligence quotient is so near the dividing line between imbeciles and morons, makes me inclined to place her in the latter class. In a few days she will be placed in the Texas institution for feebleminded women of child-bearing age. This case is especially interesting from its social aspects. Had proper examination been made of this girl's mentality during her first two years of school life, and the courts taken the proper action, she would have been put in an institution before she had an opportunity to bring a child into the world with no one to support it, and which in spite of its normal appearance, in all probability carries the defective germ plasm of its mother.

BORDERLINE CASES.

1. The red-headed American girl who had the idiot in charge, is a borderline case. Her emotional reactions are quite strong. She has considerable insight and resents any imputations of her ability or character. Had she suspected that she herself was on exhibition, she would not have come here tonight. She became pregnant at the age of twelve years and has a healthy two-year-old baby. She looks more than her fourteen years. Graded by the Binet tests, Stanford revision, her mental age is ten and one-half years and her intelligence quotient 74.5. This puts her in the borderline class, and I think her social history substantiates the diagnosis. She is on the ragged edge of legal responsibility for her actions. She accused a seventy-year-old man of being the father of her child. Although the court substantiated her accusation, the sympathy of the community is with the old man, who stoutly maintains his innocence. These borderline cases are often extremely difficult to diagnose. Some claim that such persons are fully responsible, others that they have no responsibility. I think the truth lies between. Just as they are halfway in the matter of mental ability, so are they halfway in the matter of responsibility. In other words, they do have *some* responsibility and are in a degree accountable for their acts, but not to the same degree as the clearly normal.

2. The Mexican girl is thirteen years and four months old. She is the one I referred to as having failed to pass the Form Board Test for Imbeciles. You will note that she has a rather dull stupid appearance, and some facial asymmetry. She is in low first at school and not doing very well even in that grade. However, in spite of this and the fact that she failed on one particular test which is thought by some persons to have great diagnostic value, her performance tests on the Stanford-Binet-Simon scale indicate that she has a mental age of at least ten years. I am not through studying her case yet, but I believe that her mental abilities are greater than her actual school performance indicates. Of one thing, however, I am sure, and that is her potential danger to herself and the community. Any healthy thirteen-year-old girl who is still in low first after four years' schooling, and who fails to adjust immediately all the pieces in such a simple puzzle as Knox's Imbecile Form Board is either feebleminded or maladjusted in her emotional spheres, and I doubt very much the wisdom of having her continue attending the public schools. She should be at some kind of manual labor and her training be along these lines almost to the total exclusion of purely academic learning. In other words, here is a girl who, our common sense tells us, needs the benefit of institutional training even though we are unable to definitely place her in the feeble minded class.

ORTHOPEDIC TREATMENT OF INFANTILE PARALYSIS

ROBERT G. PACKARD, M. D.

(Read at the Forty-Sixth Annual Meeting of the New Mexico Medical Society at Albuquerque, New Mexico, May 10 to 12, 1928).

Infantile paralysis, or anterior poliomyelitis, is a comparatively recent affliction throughout the world, only three outbreaks having been discovered previous to 1885. The disease first appeared as an epidemic in Scandinavia, with most of the smaller outbreaks appearing in Sweden. After 1904 it became at times most extensive in that country, with 1199 cases reported in 1905, and 5000 in 1912. In our own country the first large epidemic was in 1894, in Vermont, with the disease being endemic in that state ever since, increasing occasionally to epidemic form, as shown by the report of 300 cases in 1914. New York state contributed 2500 in 1907, and over 9000 in 1916. During the last fifteen years poliomyelitis has been recorded in every state in the union, so that now the virus is widely distributed. In this part of the country the disease has been, fortunately, sporadic. During the past two years there have appeared each fall definite waves of infantile paralysis, with the result that, during the past eighteen months, forty-three cases of poliomyelitis have been admitted to Children's Hospital in Denver.

For the purpose of description, infantile paralysis may be divided into three definite phases: the acute or febrile, the convalescent or early paralytic, and the chronic or late paralytic stage, each with its definite symptomatology and general plan of treatment. I wish today to outline especially the orthopedic treatment of the disease, merely mentioning the initial measures of therapy.

In the acute or febrile phase, manifested by fever, pain, and meningitic disturbance, and lasting for some six to eight weeks, the treatment is symbtomatic or preventive, for it must be recognized that paralysis is not an invariable feature of the disease, probably over fifty per cent of poliomyelitis cases not showing any paralysis. On account of this lack of paralysis in many cases of poliomyelitis, it is believed that innocent carriers play a large part in the spread of the disease, whether these carriers have had a mild attack or whether they are healthy persons that have associated with the sick. In the acute attack the child should be put to bed, isolated, and generally kept quiet. A quarantine period of three weeks should be insisted on. For the preventive treatment much has been written. Immune horse serum of Rosenow has been administered with probably good effect in many instances. This immune serum from the horse depends upon the injection of a cultivated polymorphous streptococcus, originally found, not in the cerebro-spinal fluid or blood, but in the brain and cord of fatal cases. There is, however, much question as to the identity of this organism as the causal factor of poliomyelitis. Flexner and Noguchi of New York, on the other hand, have succeeded in growing a minute anaerobic globoid body from poliomyelitic tissues, which, when injected intracerebrally, intraspinally, or intraperitoneally into monkeys, produces experimental poliomyelitis, and which is again obtainable from the injected monkey's central nervous system. Flexner has also shown that immune bodies are present in the blood of recovered cases. However, to date, the most practical serum to use seems to be convalescent human serum, preferably from cases having contracted the disease two to six months previously, though cases of five years' standing possess serum of some immunity. Though the literature, for the most part, advises the introduction of the serum intraspinally and intravenously, Frank Dickson of Kansas City has for the past few years been introducing either the whole blood or the blood serum intramuscularly. He advises the injection of some thirty cubic centimeters of blood, repeating the dose two or three times within twenty-four to forty-eight hours. He reports good results. However, upon the whole, there is yet no standard preventive treatment, so that much more experimental work must be done along this line. The strictly orthopedic treatment in this first stage of poliomyelitis is limited to keeping the patient flat on his back in bed, maintaining the feet at right angles with the legs, and the legs in full extension at the knees.

But with the second and third stages, the orthopedic treatment of infantile paralysis is more concerned. The second phase, or convalescent or early paralytic stage, begins with the conclusion of the fever and the end of the period of muscle tenderness and lasts until the paralysis becomes stationary, extending over a period of some two years. During this time, orthopedic treatment is, for the main part, mechanical, consisting in two general measures: the prevention of the development of deformity and the restoration of muscle power.

The majority of deformities can be, and should be, avoided. When we remember that not only is partial paralysis of a limb more common than total paralysis of a limb, but also that the individual muscles are more commonly partially paralyzed than totally paralyzed, we are reminded by the

pathologist that the reason is easily found. The nerve cells which supply the various muscles lie in th anterior horns of the cord in longitudinal columnar bundles. The muscle, then, deriving its innervation from a group of cells of several segments, loses only those centers destroyed by the transverse lesion, which is the result of the virus of poliomyelitis reaching the cord by means of the blood approaching the anterior horns by horizontal branches of the anterior spinal artery. Thus it is logical to reason that a partially paralyzed muscle may be materially helped by further development of the remaining centers, and by further development of other muscles of the same muscle group. In this way can be explained the occasional complete recovery of a paralytic case.

Deformity occurs as the result of various factors: first of all, and most important, by muscle unbalance, as when an extensor muscle is paralyzed but the opposing flexor muscle is not affected, resulting in a flexion contraction. If this original muscle unbalance is not corrected, ligamentous contraction makes the deformity more permanent. Improper attitudes in bed, as sitting up, may lead not only to various degrees of lateral curvature, a most obstinate problem, but often to flexion contraction at the hips, or at the knees if the latter are allowed to bend when pillows are placed underneath them—a most common error that we find. Then, too, early standing may promote scoliosis, while foot deformities may result from weight-bearing if certain muscles of the lower leg are involved. Gravity is another factor to be looked out for, in that it is often responsible for foot-drop. But to allow deformity to begin at this stage, in most cases is inexcusable, though it is true that the insidiousness of its progress is often responsible for its development. It is also true that, in many instances in spite of all precautionary measures, we find deformity present, as in gastrocnemius paralysis, where the contraction of the dorsiflexors of the foot has produced the calcaneus or prominent dropped heel.

The prevention of deformity, then, is the first practical consideration in the convalescent or preparalytic stage. A thorough muscle examination should be made to determine to what degree the various muscles have been affected. At the Children's Hospital in Denver a muscle chart is used on every case for the purpose of recording the amount of muscle power still remainnig. A scale from zero to five is used, so as to classify the muscle as totally paralyzed, trace, poor, fair, good, or normal. This chart is useful, both for directing the particular treatment necessary, and for comparing with later examinations. For if we are able to anticipate the character of the deformity to follow in the event of inefficient treatment, we can use our measures to counteract the particular tendency. Another method of recording the amount of paralysis is the spring balance muscle test of Lovett, of Boston, by means of which the power of muscle groups (more particularly than individual muscles) is rather accurately registered by the pull against a spring balance. To quote Lovett: "The method aims at pulling against a fixed position assumed by the patient, rather than attempting to have the patient initiate a movement." By training two persons to carry out these tests, so as to coordinate the pull of the muscle and the registration of the pull on the scales, Lovett was able to make very accurate observations and to note the progress of muscle power.

It is at this second stage of the disease that many of these cases come to our attention. The initial stage of the disease has usually not been given the significance it deserved, and the parents have found that the child is not getting over the weakness of a leg or an arm, and is, perhaps, showing some definite wasting of the limb, often complicated by some joint contraction. The child has recovered from its fever and muscle tenderness, but is still unable to use the limbs normally, and, if made to stand, is perhaps unable to balance itself without holding onto the bed, or if urged to walk, either deliberately falls down, or gets about most awkwardly. Fortunately, at this time we find the case not too far advanced to correct the beginning contraction, to prevent permanent deformity and to institute measures designed to restore muscle power. When we realize that the presence of deformity interferes with the return of muscle power and that function is naturally lessened by such contractions, we can see how important it is to treat these disabilities as early as possible, and, in some instances, even before they occur. It is our duty first of all to determine just what muscles or muscle groups are involved and what contractions are liable to occur. Records will show that the most commonly affected muscles are the gluteals, the quadriceps, the anterior tibial and dorsiflexors at the ankle, and the deltoid. The muscles of the back and the abdomen must not be overlooked. The practical measures at this stage are simple enough, the appliance of some form of brace or cast or other splint so as to keep the foot at a right angle with the leg, to keep the knee straight, and to overcome the tendency to flexion at the hip by not allowing the patient to sit up. If the ab-

dominal muscles are found weak so that the child has difficulty in coming to a rising position unaided, or if the back muscles allow a lateral curvature of the spine when the trunk is erect, the child should be kept recumbent, best accomplished by resting in a plaster shell. In the case of deltoid paralysis, abduction of the arm should be insisted upon by the simple measure of tying a bandage to the wrist and fastening it to the head of the bed. Further examination will show whether it is necessary to maintain, for instance at the ankle, the position of inversion or eversion of the foot to relax a paralyzed invertor or evertor. This is best done by the application of a short plaster of Paris cast. The question of when to let the patient up from bed is a much disputed one, depending upon the severity of the affliction, and the progress of recovery. It is my own practice to keep the child recumbent for a long period, unless the paralysis has no definite relation to weight-bearing, as in paralysis of the upper limb. It is true we have to consider the attitude of the parents who often feel we are not doing much when we insist on long bed treatment, but certainly where we have paralysis of the back or abdominal muscles, recumbency is by far the best position for the patient to assume.

We have tried to emphasize from the foregoing that the prevention of deformity is the primary practical consideration in this convalescent or pre-paralytic stage. The second general measure is best described as the restoration of muscle power or the re-education of the affected muscles. This method of treatment must not be considered apart from the efforts used for the prevention of deformity, but should be practised at the same time. Such a method includes the following therapeutic measures: heliotherapy, hydrotherapy, massage, electricity, and intelligent muscle training. Heliotherapy might be regarded as a general measure, and such it is, but we have learned that in this disease, with its tendency to muscle wasting, the rays of the sun do improve the muscle tone at the same time that they increase the skin nutrition. Hydrotherapy stimulates the circulation by the use of a warm bath to the part, followed by a very few minutes of a cold bath. It also serves another purpose, for if the child can be placed in a sufficiently large tub or pool of water to which has been added salt so as to increase the buoyancy of the water, he can better perform his exercises, now that the element of gravity has been removed, and that friction has been reduced to a minimum. Electricity has not met with much favor from most orthopedic men, and is, in general, much overrated. Probably no real scientific work has been done along this line. Massage, on the other hand, is a most valuable adjunct—its chief value being in keeping the muscles fit from the time the tenderness has disappeared until such time as they begin to function again. It should be done by a trained masseuse, especially one who knows enough not to overdo. With massage. some form of dry heat is of advantage, best supplied by some type of electric baker, or perhaps a carbon light.

Muscle training has become, of late years, almost a science in itself. It is hard to define, but can be said to consist in training the patient to perform certain motions through the stimulation of impulses from the brain by means of the remaining uninjured nerve centers to the partially paralyzed muscles. Such muscle training or muscle exercise is thus designed to accomplish the return of muscle balance in two ways: by strengthening the affected muscles themselves, and by further developing the muscles' nerve centers in the cord that have not been injured by the poliomyelitis. It is best carried out by a trained assistant who understands the anatomy of the muscular system and who is cognizant of the particular deficiencies in the given cases. The art of muscle training consists in the proper correlation of passive motion, in which the part is brought through the full arc of motion at the joint by the assistant, and the active motion by the patient, who attempts to move the part through the same arc. Of course, before any intelligent muscle training can be started, a thorough examination of the muscular system must be made and recorded. It is necessary to determine whether the muscle in question has any evidence of contraction, whether it can function against gravity, or aganist the surgeon's resistance, or, finally, whether it can perform its normal work. For instance, let us examine the gastrocnemius muscle, the normal function of which is to plantar flex the foot, and in standing, to raise the heel from the floor. The patient lies on his side on a smooth table with the foot held by the assistant at right angles to the leg. The patient is instructed to push his toes down, and we look for a contraction in the calf. If such occurs, but not enough to plantar flex the foot, we record it as "trace." If a bare plantar flexion takes place with the leg recumbent, we record "poor." If, when the leg is raised to the vertical with the patient prone, the gastrocnemius can overcome gravity, it is called "fair." Again, if the motion can be accomplished against the surgeon's resistance, we make the notation "good,"

while, if the patient can stand and raise his body weight, we record it as "normal." Having then determined just what this particular gastrocnemius is capable of doing, we can outline our muscle exercise. As suggested above, the exercises are best carried out on a smooth leather-covered table on which has been sprinkled some common dusting powder to reduce friction to a minimum. Concentration of effort is best secured by leaving the patient and the assistant alone in the room, for often the whole attention of the child is needed. Fatigue must be very carefully guarded against in each day's exericse, and occasionally a day or two of rest must be allowed if the child shows any tendency to dislike the task set down for him.

Now, in the case of the paralyzed gastrocnemius, for example, let us suppose that the child is unable, when standing, to raise the body onto the toes. He is placed upon the table, lying on the affected side, and the paralyzed limb uncovered. I believe it a very good plan to heat the limb first for a few minutes under the electric baker or carbon light before beginning the special exercises. With the foot assuming a right angle position, the patient is told to concentrate his attention in trying to plantar flex the foot. If he can not move it at all, or can only carry the foot through part of the normal range of motion, the assistant should complete the arc by passive motion, bringing the foot into complete plantar flexion, so that the patient's mental attempt will not be interrupted. When the patient is able later to move the foot through the whole arc, then resistance should be offered by the assistant, graduated from a slight resistance at the beginning to strong resistance in the middle, and back to slight resistance at the end of the movement again. As time goes on the muscle will be able to encounter more and more resistance from the assistant, and, if recovery continues, will be strong enough to raise the heel from the floor in standing. All the muscles of the body can be exercised on this same principle if their normal functions are known. Let me caution here once more against fatigue. It has been shown that too often an overzealous parent or trained aide has done more harm than good by overworking the paralyzed muscle. Judgment must be used, too, in making the exercises a joy rather than a bore, so as to get real cooperation from the child.

We have stated above that muscle training must be carried out in conjunction with the measures to prveent deformity, and we said that braces and casts were used to hold the joints in correct positions. Of course, at the time of exercises, the apparatus must be removed. In the case of a cast, we plan on making it strong enough to allow of dividing it into a front and back half. This is known as bivalving, and requires straps and buckles to hold the halves secure after replacing them. The neatest plan is to cover the casts with stockinette so as to keep them from crumbling and to keep the cotton or sheet wadding in place.

Now comes the question of when to let the poliomyelitis case walk. It is a hard question to answer. We do not want to delay recovery by putting added work on already weakened muscles, by fatiguing them unnecessarily, and we do not wish the strain of body weight to cause any existing deformity to increase. Probably, then, the best answer is that standing and walking may be allowed when the limit of improvement in bed seems to be approaching, which may be after a few weeks, or, in severe cases, after some months. At such time, further ortopedic apparatus is generally necessary to protect against increasing deformity and to help maintain balance. Various forms of splints must be used, depending upon the particular paralysis. In the case of a tendency to lateral curvature of the spine, there must be worn some type of corset, cast, celluloid or leather jacket. In the case of the cast, it is previously made with the patient in the recumbent position, the deformity being held in correction by the use of bandage traction while the plaster is being applied. The celluloid or leather jacket is made over a well-fitting plaster model made by filling the original cast. In deltoid paralysis, I believe the proper procedure is not to allow the arm to hang at the side, stretching the deltoid and relaxing the joint, but to maintain the arm at right angle abduction in an aeroplane cast or brace, the forearm being held vertical or horizontal, whichever will bring the humeral head into more snug relation with the scapula. In the lower limbs, the knees and ankles must be carefully protected against strain and deformity from body weight. The feet must not be allowed to assume malpositions, and, in the absence of quadriceps paralysis, are best protected by a simple brace consisting of a foot piece, and a single or double upright extending up to just below the knee. The foot piece of the brace, which may be tilted inward or outward to preserve inversion or eversion of the foot, should not be solid at the ankle, but had best allow some play in flexion and extension, so limited that the heelcord will not stretch in gastrocnemius paralysis, and that the dorsiflexors will remain protected when

a tendency to foot-drop exists. Of course, in very mild cases, a properly built shoe with tilting of the heel on the outer or on the inner border will be sufficient, and in some cases the raised or the lowered heel is all that need be used. For quadriceps paralysis, the most practical and yet simple contrivance is the double caliper brace, consisting of uprights extending from the shoe or the foot brace upward to the groin to end in the well known Thomas ring. The knee is held secure by a leather knee cap pressing against the patella. If flexion of the knee is desired for sitting, a drop catch may be added to allow unlocking. For gluteal paralysis, probably no satisfactory brace has been devised, and it is by far best to depend upon crutches, which, after all, serve to protect against strain and deformity in the back and the joints of the lower limbs. Lovett came to the conclusion some years ago that any case of infantile paralysis, however severe, can be made to stand and walk, provided all the deformities are held corrected, and that some power remains in at least one upper limb. He stated that, with the use of crutches, balance can be maintained because the center of gravity would be within the base of the four props if the patient would only lean forward. Walking, in the worst cases, is possible, after a fashion, the patient placing one crutch at a time slowly ahead and then dragging one leg or both, taking care not to allow the limbs to advance beyond the crutches. This he termed quadruped walking. We have proved this in many cases after correcting deformities in patients that had never stood or walked, and it is surprising how readily most children will adapt themselves to progress after they have sensed the idea of balance. It seems to me most gratifying in the treatment of infantile paralysis to get a child to help himself in getting about from one room to another after he has lain about a seemingly helpless cripple, but it can often be done by the simple measures as outlined.

But now that the child is in the ambulatory stage of treatment, let us not forget the continued necessity for muscle training. The constant use of apparatus has a tendency to atrophy, unless massage and exercise are continued. Let us remember, too, that even when apparatus is worn, it is possible for deformity to develop. The braces must be constantly watched to see that they cintinue to fit accurately, and that they are not outgrown. We have seen instances where a brace or cast designed to prevent equinus, will allow development of the dorsiflexors at the expense of the plantar flexors, and then we had to deal with a calcaneus or heel drop.

As time goes on in the ambulatory stage, the patient will often gradually discard his braces or crutches. For instance, in quadriceps weakness, the child may soon learn to maintain his balance by fully straightening, or perhaps by slightly hyperextending, his knee as is done in the case of an artificial limb. At such a stage the knee may well be rid of further knee support, thus lessening the amount of apparatus that has to be carried about. It might be added here that a slightly contracted heel cord is rather an advantage than a disadvantage in quadriceps and gluteal weakness, because, when the foot is placed flat upon the ground, the short cord serves to throw the knee backward into position of balance.

Before leaving the discussion of treatment in the convalescent stage, let me emphasize the necessity of guarding against flexion deformity at the hips. Not only is such deformity most disabling if allowed to occur, but occasionally a dislocation of the hip takes place if too much hip flexion is allowed. At these joints we do not have to fear hyperextension deformity, as may occur at the knee or ankles, because the study of the anatomy of the hips reveals the fact that hyperextension is always definitely checked by the Y-ligaments, while flexion may progress indefinitely, requiring later operative treatment if not taken care of early in the disease.

Finally, the third or late phase of poliomyelitis shows a stationary paralysis with deformities, if any, more or less fixed. This is the stage at which most children are brought to the orthopedic surgeon. This period of the disease begins about two years after the acute attack, when the initial improvement has ceased. At this time orthopedic treatment seeks to correct deformity and to improve function, and is therefore for the most part surgical. This field in surgery has been very greatly developed during the past fifteen to twenty years, so that the treatment of paralysis by surgical means has become very popular, with the result that many apparently helpless cripples have been enabled to get about on their feet, and have become, to some extent at least, more useful citizens.

Operative measures, as a general rule, should not be practised on children under seven or eight, especially as regards bone work. So often normal bone growth is interfered with, notably when the epiphyses or epiphyseal lines have been disturbed. In this way, sometimes a surgical correction in infancy will produce a more embarrassing

deformity at adolescence. Likewise, often the cutting of one contracted tendon about a joint does not overcome the contracture of other tissues, and a greater deformity develops.

As just stated, operative treatment is practised for two purposes: to correct deformity and to improve function. The correction of deformity should preced operative means for improvement of function. And we have operative procedures for correction of deformity should precede operameasures more adapted for the benefit of function. In the first group may be listed stretching and manipulation, subcutaneous tenotomy, fasciotomy and muscle stripping, tendon lengthening and shortening, and osteotomy, while the latter group includes tendon, nerve, and bone transplantation, artificial silk ligaments, partial or total astragalectomy, and joint fusions or arthrodeses.

The first surgical measure to be considered in deformitv is not open operation, but the gradual stretching for a flexed or extended limb. This is advised because it gradually corrects all of the contracted tissues rather than one or two at the expense of others. Whether the correction can be accomplished at one sitting depends upon the extent of the deformity. It is better not to stretch too much at one time, because of possible shock and pain and pressure sores. And in the flexed knee with posterior subluxation of the tibia. too vigorous straightening mav result in further subluxation. We are much in favor of the slower method of wedging casts for the correction of flexed joints. In the case of a badly flexed knee for instance. under ether, a moderate stretching of the hamstrings is carried out, then, in the position of the greatest correction, a long plaster cast, carefully padded, is applied. In two or three days, about three-quarters of the circumference of the cast is cut across the flexor surface of the joint, so as to allow. during the next few days or weeks. a forcible gradual straightening of the cast and limb. each day's gain being held by wedging of the enlarging gap with a few sections of tongue depressors or the like. Very satisfactory correction of flexed limbs can thus be accomplished with practically no discomfort to the patient.

Tenotomy was probably the first corrective operative measure in orthopedic surgery. Tenotomy of the tendo Achillis dates back to 1838, when the subcutaneous division was first done. Today, tenotomy of any tendon may be done in poliomyelitis for correction of a contracted muscle, following the paralysis of the opposing muscle, but still the tendo Achillis is the one most frequently attacked for the correction of foot-drop. In younger children, the subcutaneous method is more often practised than the open method of tendon lengthening. In the simple tenotomy the sharp tenotome is inserted through the skin along the inner border of the heel cord, the tendon cut obliquely, and the foot brought into proper position. In other cases, where the opposite deformity of calcaneus is feared or where a flail ankle might follow, the lengthening of the tendon is accomplished by the open method, the sheath being split and a double zigzag incision and suture being done. Other tendons sometimes cut are the peronei, and the hamstrings. A cast should alwavs be applied after a tenotomy, to be worn four to six weeks, preferably bivalved to permit of muscle training. Instead of tendon lengthening, sometimes a tendon shortening or reefing is done. as in the case of a calcaneus with a relaxed heel cord. As a rule. however. such a procedure is not verv successful. because the once stretched tendon is prone to stretch again.

Fasciotomv with muscle stripping, is used verv successfullv in two tvpes of deformity —the cavus or hollow foot, and the hip flexion contracture. For the cavus foot, the operation of choice has come to be known as the stripping of the os calcis, devised by Steindler of Iowa Citv. An incision is made along the inner aspect of the os calcis, exposing the plantar fascia. The fascia is divided crosswise. the small flexor muscles of the foot and the long plantar ligament are stripped off the periosteum of the os calcis, and the operation finished by wrenching the foot, which serves to reduce the deformitv. A cast should be worn six to eight weeks. In the operation for hip flexion, Soutter of Boston was the pioneer. In his method, through an incision made along the anterior edge of the tensor fasciae femoris, the fascia between the anterior superior spine of the ilium and the trochanter is first divided. then the muscles and fascia attached to the spine are stripped subperiosteally, to be dropped down toward the anterior inferior spine where they make attachment on forcible correction of the thigh flexion. This operation has been improved by Campbell of Memphis, who detaches not only the anterior superior spine, but also the anterior portion of the crest. which he implants just above the rim of the acetabulum. He also divides the short head of the quadriceps muscle. His results are excellent.

Bone operations for the correction of deformity include principally various osteotomies for extreme conditions. In a bad knock-

knee, a transverse osteotomy of the femur may be indicated. The bone is exposed just above the condyle, fractured by osteotomy and brought into correct alinement. Long standing cases of infantile paralysis, dating from early childhood, will often show an eversion of the lower leg, due to an external growth of the tibia. This objectionable deformity can be corrected by osteotomy of the tibia and fibula and forcible internal rotation. In severer rotary deformity, two osteotomies may be necessary in the tibia. At the ankle, a long-standing varus or valgus or cavus deformity may yield only to a cuneiform or wedge-shaped osteotomy, the direction of the base of the wedge depending upon the correction desired. An incision is made to expose the front part of the body of the astragalus and the astragalo-scaphoid joint. A V-shaped section of the neck and head of the astragalus, and perhaps of the scaphoid, is removed for the varus or cavus deformity with the base facing outward or upward, while for the valgus deformity the wedge removed will include some of the cuboid bone, the base of the wedge facing inward. Other corrective bone operations include fusion of the spine for paralytic scoliosis, and astragalectomy for calcaneo-valgus at the ankle, but these are more properly included among the measures for improvement of function.

Only recently another and rather radical bone operation has been used. It is a most painstaking operation, that of bone lengthening for cases of shortening of one limb. The operation is rather complicated for description and involves much accurate apparatus. Briefly, it consists in exposing practically the full length of the femur or tibia, which is divided into two longitudinal halves by a motor saw and each half cut across at the upper or lower end. This permits the sliding of one fragment on the other after each has been secured by a long steel pin drilled transversely through it and attached to overhead apparatus. The wound is closed with the pins protruding through the skin. The lengthening is accomplished slowly, at the rate of about one-sixteenth of an inch each day, so as not to stretch too rapidly the muscle, nerves, and bloodvessels. In this way, as much as two inches in length can be gained.

After deformities are corrected, the operative work for the betterment of function, deals mostly with stabilizing measures. Tendon transplantation which dates back to 1882, is first to be considered. It has not been marked with a great degree of success, though in a few joints of the body better muscle balance has been secured. This is particularly true in cases of talipes varus and valgus, and occasionally true in quadriceps paralysis. The technic in transplantation for valgus deformity will give the best description. Here the transplantation aims to replace the paralyzed anterior tibial by a healthy peroneus. It must be remembered as a general rule that any deformity must first be corrected, that the transplanted muscle must be a healthy one, and possess strength equal to the normal power of the one to be replaced. A gliding apparatus must be provided, and a straight line of pull must be established, with the end of the healthy tendon secured to bone rather than to the paralyzed muscle. Most important of all, careful after-treatment with muscle training is to be insisted upon. In the case of transplantation for paralyzed anterior tibial producing a valgus, the following procedure is carried out: A long incision on the outside of the leg and ankle exposes the peroneus longus, which is severed near its insertion, raised from its sheath, carried across the leg within the fascia, to the sheath of the anterior tibial muscle, which has been relieved of its tendon after severing of the latter at its insertion. The peroneus is then carried down the sheath of the anterior tibial and, with some tautness, tied to the denuded internal cuneiform bone, over which the periosteum is sewed. The foot is casted in position of varus. If the technic is carefully carried out, a fairly satisfactory result may be anticipated. Many other transplantations might be mentioned, as that of the common extensor of the foot and great toe into the metatarsals, for clawfoot; one hamstring or sartorious forward into the patella or patellar ligament; the tensor fasciae femoris to the patella, for quadriceps paralysis, or to the upper femur for a paralyzed gluteus medius; and the erector spinae to the posterior aspect of the femur, for gluteal paralysis. In the upper extremity, a number of transplantations have been done for contractions of the forearm, hand, and fingers.

Nerve transplantation has not been successful enough to discuss here. In bone transplantation I want to mention two outstanding examples. Many of you are familiar with the Albee bone graft for tuberculosis of the spine—the same technic is carried out for paralytic scoliosis. It consists in placing a strip of bone from the anterior aspect of the tibia across several vertebrae by inserting it between the split posterior processes, for the purpose of correcting part of the lateral flexion of the spine. Sometimes the graft of bone is placed along the concave side of the posterior processes.

Probably better than the Albee graft is the Hibbs' fusion of the spine which is accomplished by establishing five lines of fusion throughout some five or seven or nine vertebrae by curetting the interarticular facets on the right and left, and by fracturing the posterior processes at their bases so that each one comes in contact with the one below. The other outstanding example of bone transplantation is the bone block operation for foot-drop, devised by Campbell of Memphis. His plan is to insert bone into a space exposed behind the foot, bounded by the tibia above, the astragalus in front, and the os calcis below. If one-half to three-fourths of an inch is removed from the posterior aspect of the astragalus, a very adequate amount of bone can be transplanted, to block the elevation of the os calcis in foot-drop. Excellent results have been obtained.

The use of silk ligaments for the checking of foot-drop is not as popular as it was some fifteen years ago. The scheme was to inforce the ankle joint by the use of heavy strands of silk extending from the tibia to some bone in the foot, and secured by insertion into drilled holes covered over by sutured periosteum. These ligaments would be later covered over by fibrous tissue. But the operation has fallen into disrepute because often the silk would break, or become infected, or wear through the skin. Now, I believe, the operation is seldom done.

The operation of astragalectomy, worked out by Whitman of New York, has been found very valuable for stabilizing the foot in children between eight and fifteen years of age, where a bad calcaneus deformity has occurred as the result of gastrocnemius paralysis, making the patient walk entirely or nearly entirely on his heel, the front of the foot being drawn up by the contracted dorsiflexors. If properly done, the removal of the astragalus shifts the weight-bearing line to the front of the foot by displacing the tibia and fibula forward, creating the more stable position of valgus with no lateral motion, but preserving motion in plantar flexion.

Finally, let me mention the most satisfactory operative procedures in infantile paralysis. I refer to the various bone-stabilizing operations, especially those designed to correct foot instability. Stabilization of the foot is obtained by fusion of two or more bones. Though this sacrifices, to a certain extent, the flexibility of the foot, better stability is gained because, after all, joint motion is of very little value if it cannot be controlled, as in cases of bad valgus, or dangle foot, etc. Such fusion is most commonly done in the so-called "subastragalar joint" which refers to the articulation of the astragalus with the os calcis below and the scaphoid in front. It is at this joint that uncontrolled motion occurs in bad paralyses of the tibial or peroneal muscles. Muscle transplantation has not generally been found adequate in this connection, while a subastragalar arthrodesis, if properly done, is of immense value. Davis, of Philadelphia, did the pioneer work in this line. In his original operation he made two incisions, one on the outer aspect to expose the astragalo-calcaneal joint, so that he could denude the inferior surface of the astragalus and the superior surface of the os calcis, and one on the inner aspect of the ankle and foot so that he could remove the cartilage from the sustentaculum tali of the os calcis, from the head of the astragalus and from the articular surface of the scaphoid. He managed to get some backward displacement of the foot by the removal of the astragaloscaphoid joint and by freeing the lateral ligaments from the malleoli. This operation has been improved by Hoke of Atlanta, who added to the subastragalar arthrodesis by removing the head of the astragalus and replacing a part of it after a thorough backward displacement of the foot and correction of the alinement and rotation of the os calcis in relation to the leg, of course depending upon the particular deformity and paralysis present. Other operators have seen fit to improve upon these procedures. Arthrodesis may also be done in the midtarsal joints for dropping of the forefoot, where the subastragalar joint is not affected. In other parts of the body, stabilization has been accomplished by arthrodesis of the larger joints, particularly the shoulder, wrist and knee, when the relaxation of those joints has been otherwise uncontrollable.

I have tried to outline the treatment of infantile paralysis in its three stages. It is really too large a subject to cover thoroughly in the time I have taken, but I have attempted to place before you the high spots in the orthopedic plan of procedure for the various conditions as they presented. Let me emphasize, finally, that, in the first and second phases of the disease, much of the deformity can be prevented by a proper appreciation of the particular paralysis present. Function can be, in many cases, for a great part restored if we are careful to use gentle means of definite muscle exercise, and to postpone for a sufficient time the temptation to get the child onto his feet. Proper intelligent bracing must be insisted upon. In the final stage, the deformity must be properly analyzed before operative interference is undertaken and it is to be

done then only if we are convinced that improvement by other means has reached its maximum. In the operative work, the bone stabilization procedures have probably proved of most value.

DICUSSION

DR. C. F. MILLIGAN, Clafton, N. M.: I want to congratulate Dr. Packard on his very excellent paper and discuss a few points in connection with it. It is, in my opinion, only through propaganda and education that we can control this disease. I wish that this paper might be published in an early edition of the journal (Southwestern Medicine) and that it might be read by all the doctors in the southwest. There are some conditions here which we should combat. There are some physicians throughout the state who deny that we have poliomyelitis. They have various diagnoses for the cases they see and, in this way, they make it very hard for other physicians to control their cases and very difficult for the public health department to isolate and quarantine them. Then, too, some of the business men in the smaller towns are opposed to quarantine on account of poliomyelitis, because it affects their business, especially in sections where there are tourists. It hurts them if we publish the fact we have an epidemic of poliomyelitis or infantile paralysis and try to keep the children from going to the movies, to the schools, churches, etc. It keeps the tourists away and for this reason they are opposed to it and try to find some doctor who will say that such disease does not exist. This puts the general profession in disrepute. They also claim that the particular child who may be ill was not at any public gathering, and deny that there is danger through contagion. Also, in this connection, we have a good deal of trouble with chiropractors. They say the medical profession cannot cure this disease, they have no protective measures, and if a child has infantile paralysis we have very little treatment of benefit. So a lot of people think that if a child is once paralyzed, it is hopelessly paralyzed. Then along comes the chiropractor and says, "I can take care of this case," and they let the chiropractor have the child.

This paper discusses a subject which is most important, not only to the man who is treating diseases of children, but to all of us, and I hope you will help to spread this propaganda in your district. We must control this disease as much as possible and must institute proper treatment if we do have these cases.

DR. D. L. BURTON, Albuquerque, N. M.: I believe one thing which should be done in the early treatment of these cases is very important—and that is keeping the patient quiet. Oftentimes parents do not want the child quarantined and try in every way to avoid this, but if we can apply simple dressings, keep the patient in bed and absolutely quiet and comfortable, a complete recovery may occur.

DR. R. G. PACKARD (closing): Dr. Burton is right. Children are extremely benefited by quiet and rest. Children are very often sick and the parents want them to exercise—to bend their knees and ankles. It is our duty to prevent such motion—to keep the knees perfectly straight, and not to exercise the tender muscles. The tendency is to exercise them, but it is the wrong thing to do and the condition is often made much worse by massage or exercise. The muscles affected by this disease are always the tender ones. If you will only keep the child in bed, and restrain your own temptation to test out and exercise these muscles until the period of tenderness is over, you will get a much better result.

POLIOMYELITIS: ITS DIAGNOSIS AND MEDICAL TREATMENT

HENRY DIETRICH, M. D.
Los Angeles, California.

(Read before the thirty-sixth annual meeting of the Arizona State Medical Association at Tucson, Arizona, April 19-21, 1928.

An intelligent discussion of any disease will be promoted by a short resume of its pathology. This is particularly true of poliomyelitis, the pathology of which, in a striking manner, suggests to us the clinical symptoms encountered and also unfolds to us, to some extent, a rational form of treatment. The disease, when it localizes in the central nervous system, at first attacks the delicate and highly vascular piamater. The invasion continues by way of the lymph spaces of the vessel sheaths as they penetrate the cord. Hyperemia, perivascular infiltration, edema, and hemorrhage take place. We have, therefore, a lepto-meningitis. When the virus attacks the central nervous tissue proper, we again see the same reactions, hyperemia, perivascular infiltration, edema and hemorrhage. Depending upon the severity of the process, this is followed by partial or total destruction of the nerve cells, resulting in various degrees of permanent paralysis. Motor disturbances occur whenever those cells and fibers which have to do with motion are in any way affected by pathological processes. Pressure and stretching of the cells and fibers will impede their function. Depending upon the kind and degree of damage which has occurred, paresis or paralysis will follow. If the motor neurones are destroyed, motion ceases. The process of inflammation is not confined to the gray matter, but very frequently involves also the white matter and the spinal ganglia. The term poliomyelitis, polio meaning gray, is therefore a misnomer.

No matter whether we are dealing with a poliomyelitic lesion of the cord, medulla, pons or brain, the reaction in the tissues is always the same. With the triology of hyperemia, edema and hemorrhage constantly in mind, we will have little difficulty in understanding the many and varied clinical pictures which this disease presents. There is an increase in the amount of spinal fluid. It very soon becomes slightly opalescent; the cell count becomes abnormal in that the number of cells may range from thirty to forty up to two thousand. Early, and only transiently, they are the polymorphonuclear type, very soon being replaced by mononuclear cells. Chemical examination of the fluid will show the presence of globulin, a normal sugar content (fifty to seventy-five

milligrams per hundred cubic centimeters), and a normal chloride content (seven hundred to seven hundred and fifty milligrams per hundred cubic centimeters). Bacteriological examinations of smears and culture are negative.

Lesions are found in other organs also. These were not considered important as long as we regarded the presence of paralysis as requisite to the diagnosis of poliomyelitis. Today we recognize poliomyelitis as an acute, general infection in which serious involvement of the nervous system may or may not occur. In cases dying during the acute stage, there is always found an enlargement of the lymphoid tissues, especially of the mesenteric glands and Peyer's patches. Glands in any part of the body, as well as the spleen and tonsils, may show involvement. The presence of the virus has been demonstrated in the central nervous system, mesenteric and other glands, the tonsil and nasal mucosa. In 1909, Landsteiner and Popper produced poliomyelitis in the monkey by intraperitoneal injection of a suspension of the central nervous system from a human case. A few months later Flexner and Lewis reported their success in transferring the disease from monkey to monkey. They injected a suspension of poliomyelitic tissue into the brain. The etiologic factor in infantile paralysis is a living organism which multiplies in the tissues but, as yet, has not been definitely isolated. It passes through a Berkefeld filter. The disease can be produced by bringing the virus in contact with the nasal mucous membrane by means of intranasal tampons. It is believed that the virus passes directly by means of the lymphatics through the cribriform plate to the pia-arachnoid membranes. Animals once having been infected cannot successfully be re-inoculated. The blood serum of monkeys and of human beings who have recovered from the disease, contains substances which will, to some extent, protect others against the disease. The protective bodies appear in the blood on about the sixth day and can be demonstrated for twenty years or more afterward. The virus of poliomyelitis is very susceptible to drying, sunlight, hydrogen peroxide, chlorine and bichloride of mercury, but resistant to phenol. The thermal death point is about 120 degrees. This data gives us important information regarding methods of disinfection. F. R. M. Walshe very correctly states that a practical conception of the malady can be given in three clinically and pathologically distinct stages of evolution: (1) The stage of general infection. This is characterized by fever, malaise, diarrhea or constipation, irritability and behavior customary to the febrile child. (2) Stage of invasion of the sub-arachnoid space. This varies in intensity and therefore the symptoms vary. In this stage we find fever, headache, pain in the back and limbs, rigidity of spine, muscular twitching, tremor, vomiting and changes in behavior. During this stage the spinal fluid is increased. It soon assumes a slightly opalescent or groundglass appearance. The cell count will vary from thirty to forty up to one or two thousand. Early, and only transiently, polymorphonuclear cells predominate, very soon however giving place to a lymphocytosis. (3) Stage of invasion of the central nervous system proper. When the virus establishes itself in the nervous system, paralytic phenomena become established. The reflex arc is broken, resulting in absence of reflexes. The time element between the second and third stage is frequently short. It may be only a matter of hours. This should impress upon us the importance of instituting treatment early and promptly. As long as the paralysis is principally due to hyperemia and edema full restitution is still possible. It is only when the central nervous system becomes itself invaded and destruction takes place, that paralysis occurs, and it may be well here to reiterate that, in many instances in which infection has taken place in the human being, this stage does not occur, and therefore in every epidemic we have many individuals who are victims of the disease but recover without any signs of paralysis. The degree and permanence of paralysis in a given case, will depend upon the exent and thoroughness of the actual destruction occurring in the central nervous system.

Various observers and authors have given us many different clinical and pathological classifications of this disease. From a practical and clinical standpoint, I believe we can group the cases under five definite headings.

(1) The arrested or abortive type.
(2) The spinal myelitic type (flaccid paralysis).
(3) Acute bulbar type (cranial nerve paralysis).
(4) Encephalitic type (spastic hemiplegias, ataxia, etc.).
(5) Meningitic type (with or without paralysis).

1. The arrested type. This type, except during an epidemic, is probably almost invariably overlooked. It is an extremely important type because, in some epidemics, it has been estimated that this arrested form

occurred more frequently than the paralytic form. Transmission of ths dieease may at times be largely due to contact of these undiagnosed, non-paralytic patients with the remaining population. During an epidemic it is, therefore, almost imperative that we ferret out and recognize these cases in order to diminish the material upon which the disease feeds. The symptoms of onset of poliomyelitis are the symptoms of the arrested form of the disease. We will mention these here now and avoid repetition. The diagnosis will be strongly suggested by (1) headache; (2) rigidity of neck and spine. The rigidity of neck is usually in the anteroposterior direction, lateral movements are possible unless there is also involvement of the sternomastoid muscle. (3) Rapid pulse; (4) rapid respiration from no apparent cause; (5) increase in spinal fluid with slight opalescence or ground-glass appearance and a cell count usually varying from ten to three or four hundred. Globulin is increased, sugar and chlorides are normal. (6) Rise in temperature which may be of short duration and varies from 100 to 105 degrees, generally declines by crisis when the paralytic symptoms come on; (7) hyperesthesia and change in disposition, the patient may be languid or even stuporous; he may exhibit restlessness and anxiety and usually manifests a decided disinclination to be handled or moved. Hyperesthesia is an extremely frequent symptom and, at times, almost intolerable in its intensity. It is often accompanied by myalgic pain in the back, extremities or abdomen. (8) Tremor; muscular twitchings, ataxia and incoordination may be evident at a glance or, in other instances, detected by careful examination. (9) Gastrointestinal symptoms, such as vomiting, diarrhea, or constipation, usually accompany the onset. In some epidemics diarrhea seems to have predominated; in others, constipation. (10) The reflexes are never absent, but usually exaggerated during the first two stages of the disease. (11) Sweating and the occurrence of various skin eruptions might also be mentioned. Herpes is rare

Suspicion of poliomyelitis, I would say, will rest upon the presence of headache, rigidity of neck and spine, very rapid pulse and rapid respiration, hyperesthesia and fever. The examination of the spinal fluid will convert our suspicion into a certainty.

2. Spinal myolitic type. This is the type characterized by flaccid motor paralysis of the muscles supplied by the spinal nerves. Practically all paralytic cases develop their paralysis on the third day, or thereafter, of the disease; with the onset of the paralysis, the general symptoms subside or decrease in severity. The hyperesthesia of the affected parts and the pain and tenderness of the spine, are the exception to this general rule. The legs are involved more frequently than the arms. All possible combinations of paralysis of arms, legs and torso occur. With the onset of paralytic phenomena in an extremity, the reflexes become abolished. The reflex arc has been broken, due to invasion of the central nervous system by the disease. If we have, for instance, a paralysis limited to the right lower limb, the patellar reflex in that limb will be absent. The same reflex in the left limb will be normal or, in all probability, exaggerated. When paralysis has set in, it may progress for several days, then recession is the rule. Progress of paralysis after the patient has become afebrile is rarely seen. The extent of the paralysis at the onset does not allow us to come to any conclusion or make any prediction as to the amount of permanent damage done to the central nervous system. In some instances, complete recovery occurs; in others, only one or more muscle groups remain permanently ineffective; in others still, there is little or no regression of the original paralysis. Paralysis of the muscles of the torso are at first frequently over-looked; they are generally more persistent than those of the extremities. Obstinate constipation may be due to a paresis of the involuntary muscles of the bowel, together with a paralysis of the abdominal muscles. When the diaphragm becomes involved, the upper abdomen recedes during inspiration and bulges during expiration. Fixation of the chest denotes paralysis of the muscles of respiration; abdominal type of respiration is the natural consequence. We must mention here, also, the acute ascending, or Landry's, type of paralysis, which sets in with an involvement of one or more extremities and progresses upward, sometimes slowly, more often rapidly, finally involving the medulla, as evidenced by a paralysis of deglutition, and ending in death when it reaches the respiratory center. This is a very serious form of the disease; recovery may take place but the outcome is frequently fatal.

3. Bulbar type. In this type, as the name implies, there is paralysis of the muscles supplied by the nerves which take their origin in the bulb or medulla of the cord. In some epidemics, or in some stages of certain epidemics, this type has occurred frequently. In the Los Angeles epidemic, a large number of the cases fell under this heading. The facial nerve is most frequently affected, either alone or in conjunction with

other cranial nerves. A unilateral facial paralysis may be the only manifestation of the disease. Paralysis of the muscles of deglutition and of the motor muscles of the eye, is common. Dysphagia, with salivation and regurgitation of food through the nares, has many times led to a diagnosis of diphtheria. The reflexes in the bulbar type are exaggerated, not absent. The mortality rate in this type is high, due to involvement of the respiratory center in the medulla. It may be well to state here that a fatal outcome in poliomyelitis of whatever type is usually due to respiratory paralysis. Complete functional recovery in those cases which survive the illness, is much more common in the bulbar type than in the spinal form of the disease.

4. **Encephalitic type.** During every large epidemic some cases of this type are reported. At other times the diagnosis would not and probably could not be made. These cases are represented by spastic paralysis, spastic hemiplegias, athetosis, ataxia and occasionally mental defects. I mention this type merely to call attention to the possibility of a spastic form of the disease with contractures and relatively little atrophy.

5. **Meningitic type.** This is not a very common form, but it does occur frequently enough to be a diagnostic problem. The symptoms are those of a severe meningitis, frequently with convulsions. A paralysis may set in during the course of the disease and help clear up the diagnosis. A careful spinal fluid examination, only, will enable us to differentiate it from the various other forms of meningitis.

DIFFERENTIAL DIAGNOSIS

We must continually bear in mind that the disease may present an extraordinary number of clinical pictures closely resembling other infections. The disease is undoubtedly on the increase, and we may expect periodic recurrences of epidemics. Only by thinking of the possibility of poliomyelitis, whenever we encounter a patient with symptoms suggestive of involvement of the central nervous system, will we save ourselves the humiliation of overlooking this disease when it occurs sporadically or early in an epidemic.

Fully ninety-five per cent of poliomyelitis occurs in children under sixteen years of age. I will, therefore, consider only those conditions which we are called upon to differentiate during this early period of life. In the pre-paralytic stage we need to differentiate it from almost every acute infectious disease. To attempt to do so before a body of medical men would be extremely tiresome and would savor of pedagogy. Where I deem it important I will state a few points in differential diagnosis, and, in other instances will merely mention the disease.

(1) **Epidemic and suppurative meningitis.** In these conditions the nuchal rigidity is often more marked and more apt to be present on attempting both lateral and anteroposterior movements of the head. Kernig's sign is more frequently present in both legs. The mentality is usually more clouded and the stupor more profound and persistent. The temperature does not drop by crisis. The spinal fluid is more often turbid, the globulin is markedly increased, a persistent polynuclear cell count is the rule; sugar and chlorides are diminished and organisms can usually be demonstrated in the fluid.

(2) **Tuberculous meningitis.** The onset, as a rule, is gradual; however, remember that here again we have a disease which varies greatly in its early manifestations. Apathy, developing into a gradually deepening coma, in which there are no remissions, is nearly always present. In poliomyelitis the stupor, when present, is rarely so deep that the patient cannot be aroused and be made to answer questions. In tuberculous meningitis the temperature, which at first may be low, persists and usually increases as the disease progresses. An x-ray picture of the chest may disclose a miliary process in the lungs. The tuberculin test, if we use one-tenth to one milligram intra-cutaneously, will be positive. Do not be fooled by the statement in some textbooks that malnutrition is a diagnostic point in these cases. On the contrary, we see tuberculous meningitis very frequently in children whose state of nutrition is decidedly good. The spinal fluid is slightly opalescent and, on standing, a fibrin pellicle web forms. The globulin test is positive, but sugar and chlorides are apt to be low. A thorough search in the centrifuged fluid, after the web has formed, will often reveal the tubercle bacillus.

(3) **Epidemic encephalitis:** the diferential diagnosis may be impossible. These cases are more apt to occur in the early spring after the acute respiratory infections have been prevalent. Infantile paralysis is more frequent during the months of summer and early fall. The spinal fluid is not diagnostic. Textbooks will tell you how to make the diagnosis; all I can say is that perhaps the course of the disease will enable you to come to a conclusion and perhaps even then there will be room for doubt.

(4) **Syphilitic meningitis.** To aid us we must look for other signs of syphilis. The

Wassermann reaction will nearly always be positive.

(5) **Luetic pseudo-paralysis.** This may affect both upper and lower limbs and, as there is an apparent flaccid paralysis, the condition may be mistaken for poliomyelitis. It nearly always occurs in young infants. It is due to a luetic epiphysitis. There is usually a cylindrical swelling accompanied by pain and signs of inflammation at the epiphysial junction. Look for other signs of lues and, if necessary, the x-ray and Wassermann test will always enable an exact diagnosis.

(6) **Pseudo-paralysis of scurvy.** Scurvy is still with us. We see several cases every year. This condition has frequently been mistaken for infantile paralysis, due to the disability produced by the hemorrhages. The slow onset, the spongy, swollen, easily bleeding gums, the presence of ecchymoses in the skin, the painful swelling in the limbs, together with the presence of blood cells in the urine, and a characteristic x-ray picture, should save us from this error.

(7) **Pseudo-paralysis of rickets.** A rachitic myopathy occurs which may simulate paresis. Over-accentuated reflexes, the presence of other signs of rickets, and a diagnostic x-ray picture, will make the diagnosis.

(8) **Diphtheria.** A severe angina or, more frequently, a bulbar type of paralysis may temporarily lead to this erroneous diagnosis. This was true in three of the first cases which I saw during the Los Angeles epidemic. These patients had difficulty in swallowing, with some regurgitation of food through the nose. In diphtheria the paralysis comes on much later in the disease, there is usually palatal, but not pharyngeal, paralysis. Cardiac symptoms are common, and, if in doubt, a spinal fluid examination will be decisive.

(9) **Rheumatism.** This is a rarity during the first two years of life. There is often a history of previous attacks, paralysis not present, the joints are tender and swollen, the affected parts are hot; in poliomyelitis the paralyzed limb is cool and often cyanotic. During the Massachusetts epidemic of last year, three cases underwent abdominal section for a diagnosis of appendicitis. The appendix was found to be normal, but in each instance the mesenteric glands were markedly enlarged.

TREATMENT

The mortality rate in poliomyelitis has varied in different epidemics from ten to twenty-five per cent. As we learn to recognize the arrested type of the disease, these mortality figures will naturally drop to a much lower percentage. In Los Angeles, one hundred and forty-one cases were reported, with a fatal outcome in thirty-four. Nine cases received convalescent serum and seven Rosenow serum. In the New York epidemic, comprising over nine thousand cases, ninety-seven per cent occurred in children under sixteen years of age and the greatest incidence took place in children below the age of seven.

General Treatment.

(1) Isolation for twenty-one days.

(2) Absolute rest in bed. Several observers maintain that the mortality rate and extent of paralysis can be definitely increased by exertion on the part of the patient after the onset of the disease. Bed pans should be used, urinary retention should be looked for and relieved. The patient should be handled carefully and gently and should not be molested by unnecessary procedures or examinations.

(3) Proper fluid intake must be inaugurated. Children many times do not ask for water and, unless we specify definite intervals for fluid intake and a definite amount to be taken in twelve or twenty-four hours, dehydration and acidosis will complicate the disease. Where there is stupor or coma, the Murphy drip or, better still, periodic retention enemas of glucose solution are indicated. We can also, especially in the bulbar type, resort to tube feeding or we can inaugurate a Murphy drip for both fluid and food by introducing a permanent catheter through the nose, extending six to eight inches into the esophagus. If we employ this method it will be well to fill the nose with some oily substance and also to remove the tube when not in use.

(4) Elimination is of the utmost importance. Experimentally it has not been possible to produce the disease by feeding the virus to monkeys unless peristalsis was stopped by the giving of opium. Constipation is often very troublesome and must be treated promptly by the use of enemas, flushings and cathartics.

(5) Do not force the child to assume painful position. Assist him, by using pillows or pads, to facilitate his lying in the position which he prefers to assume. If necessary, use a cradle to keep the bed-clothing off the patient.

(6) Hot tubs or hot packs may be necessary for the pain and, in extreme cases, we may need to resort to codeine or morphine to quiet the patient.

(7) No known drugs have the power to influence this disease. The use of hexamethylenamine was in vogue for many years but has now been discarded.

Special Treatment.

(1) Lumbar puncture. The purpose of this procedure is threefold:

To obtain enough fluid for chemical and cytological study; to aid in the relief of pain and headache; to serve as a possible therapeutic measure. Abstraction of the fluid, which is always increased above the normal, serves to remove pressure and thereby may possibly influence osmosis to the extent of relieving some of the hyperemia and edema which are constantly present. The part which frequent early lumbar punctures may play in preventing the occurrence of paralysis or relieving it, is still a disputed question. There is good reason to believe that it has therapeutic value and therefore should be done early and, if necessary, repeated. In this connection, a report by T. Cook Smith, and Acock and Amos, of two cases of bulbar poliomyelitis with threatened death due to respiratory paralysis, is interesting. Weed and Hughson have shown that the intravenous injection of hypertonic solutions causes a prolonged and profound fall in the pressure of the cerebrospinal fluid, preceded usually by a sharp rise. These investigators had noted also an actual shrinkage in the size of the brain and spinal cord following the intravenous injections, an observation which had suggested to them that such treatment might cause an aspiration of materials from the subarachnoid space into the perivascular lymph spaces of the nervous tissue. Furthermore, pathologists had found that in poliomyelitis a marked inflammatory edema surrounds those areas of the brain and cord which are the chief seat of the injury. It seemed reasonable to suppose that the increased pressure on the nervous tissues, resulting from the development of the inflammatory processes in a closed space, was in a large part the cause of the paralyses which are present in the acute stage of the disease, but which ultimately disappear. If, at the critical moment of the disease, the pressure resulting from the inflammation could be diminished, the acute situation might successfully be passed and the life of the patient be saved. They, accordingly, in these two patients who gave definite evidence of being in a dying condition, injected forty cubic centimeters of a thirty per cent sodium chloride solution into the right femoral vein. Both children recovered.

(2) Rosenow's Serum. Rosenow regards a pleomorphic streptococcus as the specific organism of infantile paralysis. At the present time neither he nor any other investigator has furnished us with sufficient evidence to establish this as a fact. His serum is produced by injecting horses with increasing doses of the streptococcus: unless we are willing to accept the organism which he describes as the specific factor in the disease we cannot very well accept his serum as a specific serum. Rosenow has done some very splendid work and, in January, 1927, published an excellent article giving his views on the use of his serum. A good number of cases were treated and apparently successfully. The same remarks which I shall make in my comment on the use of convalescent serum can be applied to the use of his serum. At present the use of convalescent serum seems to me to be more logical and to offer more hope of success provided we can devise means of obtaining it in sufficient quantity for use in an epidemic.

(3) Convalescent Serum. Aycock, W. Lloyd and Kagan, in an article published in the Journal of Immunology in August, 1927, have the following remarks to make.

"Experimental immunization in poliomyelitis is of interest in relation to two important aspects of the disease, specific prevention or treatment, and epidemiology. The disease is nearly always accompanied by the development of a lasting immunity. Efforts to advance serum treatment have been made along two lines: to increase the penetration of serum into the central nervous system, and to produce a hyperimmune serum. In connection with the latter, many attempts at artificial immunization have been made. Evidence from many laboratories has shown that killed virus has no immunizing power. Strains of the virus of poliomyelitis have been observed to vary in strength. However, efforts to cause such alterations in virulence artificially have been unsuccessful. That attenuation might be accomplished is suggested by the occasional prolongation of the incubation period after subjecting virus to the action of convalescent serum, filtration, or treatment with various chemical agents. On account of the analogies between the virus of poliomyelitis and that of rabies, attenuation of the virus of polioymelitis by drying has eagerly been sought. With the virus of poliomyelitis the lack of a variety of susceptible species has not permitted the use of the undoubtedly advantageous fixed virus. The authors vaccinated monkeys with spinal cords of active cases of the experimental disease dried over caustic potash. About twenty-five per cent of animals vaccinated with attenuated virus resisted subsequent intracerebral inoculation. Infection resulted in twenty-five per cent of the animals treated. Phenolized virus and those treat-

ed with glycerin dilutions, possessed little less infectivity than active virus when injected subcutaneously. A large number of intracutaneous injections of active virus does not produce infection. However, if this is repeated for a considerable time, monkeys are protected from intracerebral inoculation or the incubation period is prolonged. Scarification with virus produced no local reaction, did not cause infection and did not protect against intracerebral inoculation. Blood serum of monkeys vaccinated by the intracutaneous method neutralizes the virus of polioymelitis."

Human convalescent serum in dilution of one to sixteen has protected monkeys against the experimental disease. It is interesting to note that it has never been possible to produce the disease in animals by the injection of spinal fluid from an acute case of poliomyelitis. What I will have to say regarding the application of convalescent serum has come to me through a personal communication by a member of the Harvard Polio Commission whose report on the epidemic of last year has not as yet been published. The technic elaborated and followed by them during the Massachusetts epidemic will be given here.

The serum was obtained from any available case of infantile paralysis of any type, including the arrested type. The immunologic bodies persist in the blood of patients for many, many years. The Harvard Commission used the serum from one patient who was affected forty years ago. His serum was tested by animal experiment and found to have retained its curative properties. They prefer to use the serum from patients in whom the termination of the disease dates back six weeks to five years. There was not enough serum available for use in all cases, therefore they decided to confine treatment to non-paralytic cases only. The earlier in the disease the serum is given, the better the result. A spinal puncture is performed and the fluid drained off. Fifteen cubic centimeters of the serum is then injected intrathecally and twenty cubic centimeters intravenously. The intraspinal injection is frequently followed by signs of increased meningeal irritation, stiffness of neck, Kernig, etc., and on the following day the spinal fluid shows an increase in the cell count. Twenty-four hours later the injections are repeated. After this, only the toxic cases received more serum. They probably would have given more serum to those cases which still showed febrile reactions after this period if serum had been available in sufficient quantities. They never made use of the intramuscular or intra-peritoneal route in administering serum. The late Dr. Fleishner, of San Francisco, gave serum intramuscularly. Ninety per cent of the cases receiving serum on the first day escaped without paralysis, and only about one-third of the total cases were followed by paralytic phenomena. The use of convalescent immune serum is a definitely logical therapeutic measure and should be employed in every case of poliomyelitis until we have proved its efficiency or its lack of curative power. It undoubtedly has curative value, but its application is naturally impeded by the difficulty of obtaining enough human serum to treat both pre-paralytic and paralytic cases during an epidemic. Convalescent serum has now been used in a small way for the past ten or twelve years.

COMMENT

There are several points to be considered before we arrive at a definite conclusion as to the value of serum treatment in this disease which shows such great variations in symptomatology and incidence of paralysis.

1. An exact dosage of the serum has not been established.

2. We have no method or syndrome of symptoms before paralysis sets in which will enable us to predict whether paralysis will or will not occur.

3. We also have no way of determining after paralysis has set in how much regression the individual patient will show even if not treated.

4. Due to the greater prevalence of poliomyelitis and the many contributions on the subject, especially those calling attention to the prevalence in every epidemic of cases not developing paralysis, the diagnosis is now much more frequently made in the pre-paralytic stage. Years ago a diagnosis of infantile paralysis without evidence of motor neuron destruction would have been ridiculed.

With all the effort which is at the present time being brought to bear on the study of the disease, it seems to me possible that some method of immunization may be discovered, even though the specific organism has not been isolated.

ORTHOPEDIC AFTER-CARE OF INFANTILE PARALYSIS

JOHN C. WILSON, M. D.
Los Angeles, California

(Read before the Arizona State Medical Association at its thirty-seventh annhal meeting, in Tncson, Ariz., April 19-20, 1928).

A discussion of the treatment of infantile paralysis may be divided into three stages—the acute, convalescent and chronic. The chronic period is subdivided into an operative and a non-operative phase.

ACUTE STAGE

The acute stage is characterized by tenderness over the involved muscle groups. The extent of paralysis is more apparent than real, because of the pain associated with motion. The layman feels a positive impulse to begin exercise and massage during this period, and it will tax the ingenuity of the medical supervisor to procure complete rest during the interval. Mechanical stimulation added to irritated nerve endings, does not benefit an inflammatory process of the spinal cord and its coverings; in fact, it may permanently limit recovery.

Heat, in the form of hot salt baths, is beneficial and pleasing to the patient. This may be accomplished by lifting the afflicted person into a small bathtub or lowering the individual into the bath in a sheet. The temperature of the water should be equivalent to body heat, and may be gradually increased up to 100 to 105 degrees, depending upon the vigor of the patient. The time of immersion must be regulated according to the particular case. It is well to begin with five minutes and gradually increase the time to eighteen or twenty-two minutes daily.

Deformities develop even in the acute stage and one must be on guard to prevent them. Loss of contractibility of paralyzed muscles causes contractures of the opposing muscle groups. Deformities are chiefly due to the following causes:

(1) Foot drop, because of inadequate splinting, or pressure from bed clothes.

(2) Flexion of the knees, resulting from pillows under the knees, or inadequate splinting of the extremities in extension.

(3) Flexion contractures of the hips occur because the patient is allowed to lie in a semi-reclining position upon a bed which is not firm, with support under the knees.

(4) Adduction contracture of the shoulder is caused by failure to splint the arm in abduction and in internal or external rotation, depending upon the mucsle groups involved.

(5) Scoliosis, or lateral curvature of the spine is due to an inadequate period of recumbency on a Bradford frame or in a shell on a firm bed, and lack of a properly fitted corset when the upright position is resumed.

The character of the splints used in this phase of the disease is not essential. Bivalved plaster-of-Paris casts, plaster beds, Bradford frames or wire splints are of equal merit, if properly used.

Instances may be encountered where the tenderness is so acute that the patient is comfortable only with the extremities in a position of deformity. Fortunately, such occasions are not common. As a rule, the pain will subside sufficiently during the first week or ten days to allow splinting. Fixed deformities will not occur during this time.

CONVALESCENT STAGE

With the disappearance of tenderness one may take stock of the individual case and make a careful examination to determine the extent of the paralysis. This examination is of the utmost importance, and should be undertaken in a systematic manner. It is well to begin with the muscles of the feet and go over the entire system. A hot saline bath is often an aid in this examination. The patient will contract the weakened muscles with greater ease. An accurate record should be made at this time; a chart upon which are listed the muscles or muscle groups of each extremity, with their particular movements, will suffice.

In patients who are able to walk, the type of gait is a source of valuable information. Paralysis of the gluteus medius produces a characteristic limp. This muscle acts as an abductor of the leg, and, during weight-bearing, elevates the opposite side of the pelvis. When the patient bears weight on the affected limb, the pelvis sags toward the same side in an effort to balance, giving the impression of a short leg.

When the hamstrings are strong and the quadriceps paralyzed, the patient may walk with the knee in hyperextension. The patient will find it necessary to externally rotate the thigh or support the knee with the hand when both flexors and extensors of the leg are inactive.

In case of a foot drop, the leg is lifted high to allow the foot to clear the floor. A gastrocnemius paralysis allows the heel to strike the floor first, eliminating the spring from the step. This causes shuffling of the foot.

REST

The patient should be kept absolutely recumbent upon a firm bed during the acute stage. A soft mattress and sagging bed are not permissible because of the danger of hip contractures and lateral curvatures of the spine. If the lower extremeties are involved, the feet should be protected from the weight of bed clothes. Application of splints is not always advisable in the acute stage,

because of the added discomfort. An early attempt should be made to support the feet in case of flexor paralysis and to hold the arm in abduction in deltoid weakness.

The length of time that the patient is to remain in bed is an important phase of this problem. It is safe to say that the error is always on the side of early activity. The extent of the paralysis is a deciding factor. It may be safely stated that cases of severe double lower extremity paralysis should not be ambulatory for at least one year from the onset of the disease. The individuals may, of course, sit up in rolling chairs and be taken about after the first three or four months, but should under no conditions be allowed on their feet.

Paralysis of the abdominal and trunk muscles requires a longer period of recumbency. When the paralysis has been extensive there is no dependable apparatus which will give support to the muscles of the torso in the upright position.

Overstretching and fatigue of weakened muscles must not occur. Many weeks may be required to recover from a few hours of over-indulgence in muscular exercise.

PHYSICAL THERAPY

Massage. The chief benefit from massage lies in the stimulation of the circulation. Mechanical emptying of engorged lymphatic spaces, with acceleration of the blood flow to and from the heart, will improve the nutrition of the tissues. To be effective, massage must be carefully carried out with a distinct objective in view. Too vigorous or rough manipulation is not to be permitted.

Heat. Muscles respond more readily to exercise when the local temperature is increased. The character of the heat is immaterial. A greater amount of dry heat can be tolerated, consequently its use is more prevalent.

Electricity. There is great difference of opinion in respect to the use of electricity. When it is impossible to obtain contractions by other means, the faradic current is useful. New paths for efferent impulses around damaged nerve centers may be developed by the application of the electrodes over motor points. Much of the unpleasantness of the faradic current has been eliminated by the Bristow coil.

Muscle Training. Muscle training has two distinct objectives: First, the development of new paths over which motor impulses may travel from the brain to the muscle end-plates, circumventing damaged nerve tissue; and, second, the development of weakened muscle fibers to a state of functional usefulness. It is well to move the extremity passively through the arc of movement of the paralyzed muscle before an attempt is made to obtain a contraction. If the muscle does not have the power to carry the extremity through the full range of motion of the joint, it should be assisted. As muscle strength increases, the exercises are carried out against gravity and later with resistance.

Clear and concise knowledge of the extent of the paralysis must precede the institution of muscle building. There is nothing more detrimental to the recovery of an extremity than harum-scarum exercises. A detailed discussion of the maneuvers for particular parts of the body is not essential. If the fundamental principles of muscle training are kept in mind, any necessary group of exercises can be worked out with the aid of a little knowledge of anatomy and muscle physiology.

Under-Water Exercises. The tank, or under-water gymnasium, is being utilized with considerable success in the treatment of infantile paralysis. The warm water improves the circulation of the paralyzed muscles and the buoyancy of the water aids in active movements of the extremities. This sort of treatment is possible only in institutions, or in private swimming pools, in which water is heated. The importance of this type of exercise is so well recognized that a built-in tank has been added to the new physical therapy department of the Los Angeles Childrens' Hospital. A tank should be a part of the equipment of any center where extensive treatment of poliomyelitis is carried out.

Braces. Braces are applied to make walking possible, or to facilitate walking and prevent deformities. Braces have no beneficial effect upon the paralyzed muscles; the constriction required to secure them to the extremities impairs circulation and theoretically impedes repair. The benefits, however, derived from locomotion, counteract the untoward effects of fixation.

Braces for the lower extremities and trunk are of importance in aiding locomotion. Instability of the knee, due to quadriceps femoris weakness is the chief condition requiring brace fixation. Patients may learn to walk without apparatus when quadriceps paralysis is present in one leg, by hyperextending and locking the knee. If the weakness is bilateral, braces are essential. The long caliper splints, with or without locks, are commonly used. Foot drop can be overcome by braces with ankle joints which do not allow motion of the foot below a right angle. The reverse is true in calcaneus deformities.

Paralyzed trunk muscles are best supported by reinforced front-laced canvas corsets which extend from the trochanters to the axillae. The number and location of the steel ribs depend upon the extent of the paralysis.

It may safely be said that any patient with two good arms can be taught to walk with crutches even though the lower extremities and trunk are severely paralyzed. The crutches form two legs of the tripod, and the paralyzed extremities the third. Th patient may either swing the body forward between the crutches or hunch one crutch and leg forward alternately.

Aeroplane splints are applied to the upper extremities to prevent deformities by holding weakened muscles in a position of relaxation; they are worn both night and day as long as required. Night splints are necessary to prevent undue muscular strain and deformities. Posterior wire splints are commonly used to prevent foot drop and knee flexion.

Late Surgical Treatment. Until the necessity for specialized surgical care arises, the patient suffering from infantile paralysis may be handled successfully by any well-trained medical man who will take the time to examine his patient carefully and obtain a clear conception of the problem. On the other hand, the surgical correction of deformities and other operative procedures which are undertaken to improve function, belong to the realm of the especially trained surgeon. Time does not permit a detailed discussion of the indications for, and the technic of, the various corrective operations. Certain fundamental principles referable to the mechanics of rehabilitating measures are worthy of some consideration.

Turning to joint and muscle physiology, it is noted that, in the normal individual, the muscles controlling movement of a joint are in such a state of balance that fixation may be obtained at any point in the arc of motion which will withstand varying degrees of strain. As a general rule, the nearer a straight angle is approached the greater may be the load. If, by chance, one or more of the stabilizing forces are eliminated, the stability of the joint will be diminished. This instability can be corrected only by re-establishing the stabilizing force or eliminating the point of motion.

These principles form the basis for all sound stabilizing operations upon the feet. The feet allow four distinct movements: flexion, extension, eversion and inversion, and their various combinations. For example, paralysis of the peroneal group of muscles will result in an unstable inverted foot. The treatment resolves itself into a tendon transplant to replace the weakened muscles, or elimination of the joint which allows inversion of the foot, with balancing of the remaining forces. Ankylosis of the astragalus and os calcis will fix the point of motion and transplantation of the tibialis anticus to the center of the foot and the tibialis posticus into the tendo Achillis will balance the muscle pull. Function of the foot will then be improved without the patient being aware of the loss of motion in the subastragaloid joint.

Sacrifice of certain movements is justifiable to improve the function of the extremities. Fusion of the knee may be preferable to wearing a brace for life. This would be advantageous in an individual, such as a craftsman working at a bench, whose livelihood depended upon a stable weight-bearing leg.

Tendon transplantation in the lower extremities is usually associated with some of the stabilizing operations. Great care must be exercised to balance muscular forces after stabilization, in order to prevent the development of fixed deformities.

Provided the inner hamstring group is strong, transplantation of the biceps femoris forward, in case of quadriceps femoris weakness, gives a gratifying result.

The tensor fascia femoris may be substituted for the gluteus medius by changing its insertion to the great trochanter. Walking on a level is improved and the limp due to tilting of the pelvis disappears, while walking up stairs is rendered more difficult.

Tendon transplantation is often practical in paralysis of the upper extremity. The trapezius may be substituted for the deltoid, and the carpal flexors or extensors utilized as flexors and extensors of the fingers after fixation of the wrist joint.

Paralytic scoliosis is successfully managed only by fusion of the primary curve. A surprising amount of correction of old scoliotic deformities can be obtained by traction jackets. After the maximum amount of correction, the spine should be fused. If the scoliosis is allowed to progress until rotation of the vertebrae has occurred, marked deformities of the thorax are inevitable. Correction of the spinal curve has very little effect upon the contour of the ribs, in adolescence, while the improvement in the configuration of the chest may be striking in young children.

Leg Lengthening Operations. Shortening of the extremities is a frequent aftermath of poliomyelitis. While it is of little conse-

quence in the arms, it is of great importance in the legs.

Osgood and others at the Boston Children's Hospital, have made the observation that a sequestrum lying in contact with the epiphysis of a long bone stimulates growth of that bone. Acting upon this principle, ivory pegs were introduced into the epiphyses of the bones of the leg with rather startling increase in length of the extremity.

The femur may be lengthened about three inches by a "Z" or an oblique osteotomy and skeletal traction. Various elaborate mechanical devices are recommended to obtain this traction, but the unjointed Steinman pin is simple and practical. While there is no objection to pegging epiphyses in young children, the plastic operations on long bones are not recommended during the growing period. One can not be certain of the amount of permanent shortening before the eighteenth year.

It is hoped that this will be construed as an effort to renew interest in the various steps of the treatment of infantile paralysis, rather than to enter into a technical discussion of any particular phase.

An effort should be made to establish centers in this great expansive state of Arizona, to which children from any station in society might be brought for examination and proper treatment. This could well be done under the direction of your State Board of Health. The expenditure required for the work would be justified by the economic benefit derived during the present generation.

EPIGASTRIC PAIN WITH CONFLICTING SYMPTOMS

(Cabot's Case History No. 14162, New England Journal of Medicine, June 7, 1928).

Discussions by selected team from Maricopa County Medical Society at Phoenix, and by Group 2 of the Yavapai County Medical Society and Officers of U. S. Veterans' Hospital at Whipple, Ariz. Clinical histories were published in our September issue (q.v.).

Yuma County Medical Society

On October 9th, the Yuma County Medical Society discussed this case and have submitted the following individual diagnostic conclusions:-

Dr. Knotts:- Malignancy of head of pancreas.

Dr. Shields:- Carcinoma of body of stomach, with secondary involvement of liver and spleen.

Dr. Ketcherside:- Carcinoma of gallbladder and liver.

Dr. Cain:- Hypertrophic cirrhosis of liver.

Dr. Reese:- Gallstone disease; endocarditis.

Discussion by Maricopa County Medical Soicety

DR. HOWELL RANDOLPH, PHOENIX.
DR. T. W. WOODMAN, PHOENIX.
DR. J. D. HAMER, PHOENIX.

(Dr. Randolph)

Upon cursory examination, it would seem that the abdominal symptoms are most prominent in this case. The chief complaint, "pain and gas on the stomach," together with persistent symptoms referred to the abdomen, jaundice, clay colored stools, an indefinite epigastric mass, all point to an abdominal intra-abdominal condition.

However, there is another group of symptoms and signs which are evidence of definite trouble in the cardio-vascular system: shortness of breath during the past year: palpitation for three months; high blood pressure; a systolic murmur widely transmitted, the first sound not wholly obliterated, with an occasional extra systole: enlargement of the spleen. There is evidently some degree of cardiac embarrassment.

While the shortness of breath was said to have grown progressively more marked during the past year, there was no note of it at the initial examination. Palpitation was said to have been present for three months. The term palpitation should be used rather guardedly, as too frequently this term is not understood by the patient (unless he has suffered from it). In history taking a more useful term or phrase is "heart consciousness" of varying degrees.

The actual pathology of the heart condition is very difficult to make out definitely, because of lack of data and of certain inconsistencies in the findings. Here we have a systolic murmur at the apex, transmitted widely. That would naturally point to a lesion of the mitral valve, or possibly but very improbably with the description of sounds at that area, of the aortic valve. With an aortic stenosis, the transmission of the murmur would be in the direction of the great vessels. A glance at the blood pressure immediately rules out both of these lesions as very improbable. A blood pressure of 214/100 is incompatible with a leakage at the mitral valve. With a pulse pressure of 114 we

must have an enormous forward movement of the blood during systole, whereas if the mitral valve were incompetent to any marked degree, with the blood moving both back into the auricle and forward into the great vessels with systole, the pulse pressure as a rule is not greater than thirty or forty. It is very seldom as high as sixty. With a pulse pressure over 100 it is difficult to think of any pathological condition other than aortic insufficiency as a cause (except in marked elevation of both systolic and diastolic pressure). When blood pressure is taken under conditions of strain, the pulse varies considerably from that taken in the same patient under conditions of rest, which may account for the drop in pulse pressure to 74.

And yet, an aortic insufficiency is hardly conceivable in this case in the absence of all the signs, including diastolic murmur.

In cases of hypertension there is not infrequently heard a systolic murmur of the nature of that found in this case. It is interpreted to mean a relative incompetence of the mitral due to enlargement of the mitral ring, and it would seem that this was the most likely explanation for the murmur in this case. The hypertension here is probably on an arteriosclerotic basis, with a low grade renal damage. It is unfortunate that we have no report of urine examination, which I should expect to show a small amount of albumin with casts. There would probably be an elevation of the blood nitrogen with a definite reduction in kidney function.

The loss of weight was very gradual from about the time the hypertension was probably established four years before up to five months before her last visit. In the last five months 36½ pounds were lost. This can conceivably be accounted for by a functional disturbance of the gastro-intestinal tract secondary to the cardiorenal complex.

A crucial point in the differential diagnosis in this case is the presence of jaundice. We have here two definite attacks, one of them observed, associated, it was thought at one time, with clay colored stools. If that is reliable, there must be an obstructive type of jaundice. Without the history of clay colored stools, chronic passive congestion of the liver might be considered as a possible cause of the jaundice. There is marked splenic enlargement, but no notation as to the size of the liver. Advanced mitral valve lesions cause jaundice from chronic passive congestion at times, but there are usually signs of congestion of the lung as well.

Summing up the case from the cardiorenal standpoint, it would seem probable that we should find myocardial changes toward fibrosis, with hypertrophy and dilatation of the left ventricle, and dilatation of the mitral ring to small degree. Arteriosclerosis. Arteriosclerotic changes in the kidney. Chronic passive congestion.

(Dr. Woodman)

From the standpoint of surgical differential diagnosis we have in this woman a patient with severe pain in the epigastrium diffusely radiating, associated with jaundice and a marked loss in weight, a palpable mass close to the mid-line in the right upper quadrant, and, according to the x-ray, an irritable, rapidly emptying stomach. The conditions surgically that must be differentiated in this case, I believe, are cholelithiasis, carcinoma of the gall bladder, carcinoma of the head of the pancreas, other neoplasms of the pancreas, cyst of the pancreas, pyloric tumors and ductus choledochus tumors.

Cholelithiasis could fit in with this clinical picture. The jaundice is recurrent and does not persist. This would suggest a stone in the common duct even though the pain is not radiating to the right scapula as it does in the classical case. A negative Graham test does not necessarily mean that stones are not present, although as a general rule we are able to demonstrate them by the Graham method. Dr. Sweek and I operated on a man of this type last year. The Graham test was negative, but when we opened the abdomen we found several hundred small stones packed into the gall bladder, the hepatic, cystic and common ducts, and also in a pocket in the liver substance. The clay colored stools certainly mean obstruction of the common duct from some source.

Carcinoma of the gall bladder with stone cannot be ruled out in this patient. Malignancy is suggested by the marked loss in weight even though the red blood count has not diminished from the normal. We would expect however that in a case of malignancy of this long standing there would be some secondary anemia.

In carcinoma of the head of the pancreas the jaundice is progressive and rarely subsides as it did in this case. Pain is constant and vomiting is frequent.

In pancreatic neoplasm the gall bladder is usually enlarged and not tender. Such an enlargement would have shown in the x-ray when the Graham test was made.

A pyloric tumor usually changes with changes in posture and respiration and also is spontaneously mobile. Negative x-ray does not necessarily rule this type of tumor out but there would certainly be apt to be sufficient spasm of the pylorus or sufficient encroachment on the lumen to be detected by x-ray.

Ductus choledochus tumors would give a more progressive and more persistent type of jaundice as the tumor grew and caused more and more encroachment on the lumen of the duct.

A pancreatic cyst, benign, specific or inflammatory tumor of the pancreas would give progressive jaundice and not intermittent as was present in this case.

Carcinoma of the stomach could—with metastases to the liver—give this picture. The jaundice in such a situation, however, would be apt to be progressive, if present. Such a tumor should be easily palpated in a woman as thin as this patient undoubtedly was. Then too we would expect a secondary anemia which was not present in this case.

In conclusion, I would say that the most logical diagnosis on a basis of the history and clinical findings from a surgical point of view would have been cholelithiasis with a ball-valve obstruction in the common duct; this despite the fact that the Graham test was reported negative. Probably malignancy was starting in the gall bladder.

Certainly, despite the fact that this patient constituted what is generally considered a very poor surgical risk, I believe that no harm would have been done if an exploratory incision had been made in the right upper quadrant, close to the mid-line, under local anesthesia.

(Dr. Hamer)

This case is rather obscure, both from standpoint of the findings relative to the vascular system, and to the gastro-intestinal system. There is nothing in the patient's past history to suggest a diagnosis, or lead to a diagnosis, although the patient does date the onset of her trouble back to an attack of "gas and pain" following a hysterectomy thirteen years previously. The case record does not tell us the exact time of the attack in relation to her operation, but it is probable that it was associated with her post-operative course and has nothing to do with her present illness.

From the laboratory, we have a red count, white count and hemoglobin within normal limits, an important point to remember in this case because the lack of an anemia would tend to discourage a thought about malignancy, at least any type of malignancy which had reached any degree of progress. The Graham test and x-ray findings are essentially negative. These are important and can prove of assistance, but here they do not; however, it must be remembered that the visibility of calculi do not prove that they are the cause of symptoms, for they may be latent.

From the complaints and physical examination, one is suspicious of grave pathological disturbances in the upper abdominal region, and in a differential diagnosis one has to consider all diseases accompanied by pain in this region, of a chronic and recurring nature. To me, the presence of jaundice and clay colored stools indicate obstruction in the common duct, and I hardly think it necessary to dwell upon lesions of the stomach or duodenum, unless it would be malignancy metastasized to the liver or biliary passages; but the time element and lack of anemia rule this out.

I think this is a case of gallstones, causing partial obstruction, with symptoms due partly to mechanical factors and partly to infection. The jaundice in this condition is of varying intensity, but, as a rule, not deep. From time to time, one gets attacks of pain, the jaundice deepens, and fever rises, if the attack lasts any length of time. The gall-bladder is usually not large, the spleen is usually palpable, the stools vary in color and are generally paler than normal, emaciation is common and often attains a degree that is suggestive of cancer. This patient no doubt had an enlarged spleen, because a palpable spleen usually means an enlarged one. Was there an associated enlarged liver? The records do not state. I take it though, that if the liver had been enlarged, it would have been noted. Apparently, too, there was no ascites, so one can decide with reasonable certainty that he is not dealing with a lesion causing portal or venous obstruction.

A neurosis had been diagnosed in this case, but one must be very cautious here. Hysteria may be accompanied by pain suggestive of gallstones, but the irregularity of attacks, the presence of stigmata of hysteria and absence of localized tenderness, point the way to a true diagnosis. An hysterical individual may be afflicted with gallstones. From gastralgia or neuralgia of the stomach, (a condition of paroxysmal attacks of pain) one should seek a further diagnosis, because this condition is rare and even questionable as to its existence. Riedel estimates that 97 per cent

of these cases are due to gallstones. But the presence of jaundice here rules out a simple uncomplicated stomach disorder.

One must consider congenital adhesions. As Niles, in 1925, points out, this is a fairly common condition, especially in women, and may give rise to attacks of pain or to symptoms of neurasthenia, with indigestion. The diagnosis rests chiefly with the x-ray, which shows a pylorus further to the right than normal and very high, the apex of the duodenum being also high and fixed.

One should always remember to think of tabetic crises in these cases. However, this condition is unlikely here, although there is no data to rule it out, except jaundice.

Dr. Woodman has touched upon the possibility of pancreatic lesions, so I will not elaborate. It must be remembered that chronic pancreatitis is, in many instances, a complication of gallstones. Its recognition, however, before operation, is usually impossible. An enlarged pancreatic head is seldom palpable, but in rare instances it presses upon the common duct and causes jaundice, with a dilated gall-bladder; this syndrome suggests malignancy. Carcinoma of the gall bladder in its early stages, often resembles cholelithiasis, with which, indeed, it is sometimes associated. If carcinoma is present here, it is so early that deep jaundice, metastases and cachexia, are lacking.

Dr. Randolph has given us his conception of the possible diagnosis with reference to the vascular system. Should one consider angina pectoris or coronary thrombosis in the light of this patient's findings? Possibly so. Angina may be simulated by atypical biliary colic or vice versa. And in angina sometimes the pain may be confined to the epigastrium. Usually, though, other findings of angina suffice to make a diagnosis, if sought. Coronary thrombosis may simulate upper abdominal lesions and should be kept in mind in every case of upper abdominal pain in a person over 40 or 45 years of age. A history of previous heart trouble, hypertension and recurrent stomach trouble is significant. But here we have no basal rales in the chest, no enlarged liver, no friction rub over the precordium, no low blood pressure, as suggestive of coronary occlusion or thrombosis.

Diagnosis:—Cholelithiasis.

DR. WATKINS:—There is evidently some confusion about the significance of the Graham dye test. The fact that the test was "negative" has a definite significance in suggesting a pathological organ. The gall bladder which does not outline with the dye is now considered a diseased organ, with certain possibilities for error. If the test is repeated a second time, with the same "negative" results, we do not hesitate to give a "positive" report of pathology in the bile tract. It means either that the gall-bladder cannot fill with dye impregnated bile because of obstruction, or that it cannot fill because of inability to empty the bile which is already in it. In either event, there is a positive indication of disease.

Discussion by Yavapai County Fort Whipple Group

DR. H. T. SOUTHWORTH, Prescott
DR. GEORGE BASSETT, Whipple
DR. W. E. McWHIRT, Whipple

(Dr. Southworth)

Very briefly our case is one who reported four times to the out patient department during a period of eight weeks. The outstanding symptoms were loss of weight, digestive disturbance, jaundice, a palpable mass in the abdomen, and heart with palpable spleen.

Less briefly our case is a woman of 58 years who had lost considerable weight during the past four years, particularly the past six months; fifty-six pounds in all, twenty-eight in the past six months.

One year ago she developed a digestive upset, gas and pain relieved by soda or eating a cracker. Ten days ago she noticed slight jaundice with fever; also one year ago she developed a marked shortness of breath, persistent to present time, and which became worse.

Physical examination shows a woman with hypertrophied and infected tonsils, pyorrhea and carious teeth, heart not enlarged but has a loud systolic murmur widely disseminated, palpable spleen and a mass in the right upper quadrant near the mid line.

Hemoglobin was 80 per cent, reds 4,500,000.

X-ray of stomach showed no lesion, but a slightly sluggish peristaltic wave; duodenal cap irritable and empties rapidly. Gall bladder reported negative.

Progress of case: ten days after this she reported that she was free from pain. In the meantime the pyorrhea and carious teeth had been extracted. In two weeks she reported again still better. At this time the blood pressure was 214/100.

Four weeks after, which is the end of the eight week period, she reported that one week before she had had a very severe digestive disturbance; she had gas, was always hungry, sharp pains in the stomach and burning in the back; and one day she

had rather marked jaundice. Blood pressure at this time was 176/104; weight was 98½ pounds, a loss of eight more pounds during eight weeks.

She was told to report again in one week but failed to do so.

(Dr. Bassett)

A brief and very comprehensive picture of this woman has been given. We must take into consideration a few things: she had a bad heart, bad kidneys, high blood pressure, infected teeth, pyorrhea, bad tonsils, also pain in the stomach. Was the stomach trouble a result of these multiple conditions? There was some diffuse pain at times. The history states that a slight tumor mass was felt near the mid line, upper right quadrant. Under ordinary conditions that palpable mass may not have been palpable at all. This woman had lost nearly sixty pounds and was far from fat; it would not take much of a tumor to be palpable. X-ray of the gastro-intestinal tract was negative except for rather sluggish peristalsis and a little irritability of the pylorus. Barium enema check was negative. There could not have been cancer for any length of time without showing in the abdomen and the effects appearing in the lower extremities. She had two rather general attacks of jaundice;—the gall bladder examination apparently was normal. Considering disease of the pancreas, we expect a certain number of symptoms, the most prominent of which is vomiting and failure of digestion. There was no vomiting except one time when it was induced. There was diarrhea. One condition which we might expect is ulcer of the stomach, of which also one of the most prominent symptoms is vomiting. In a study of over fifty cases of gastric ulcer, hemorrhage occurred in practically all cases at some time. In our case there is no record of hemorrhage. The attacks were reported as rather irregular, which is not true in ulcer of the stomach, the attacks being rather prolonged over a considerable period, especially in one who has not consulted medical advice or been put on a diet. She got relief from these indigestion attacks by taking a little soda or anything to neutralize the lactic acid, or tendency for food to stay in the stomach instead of passing on down. Further along, in the duodenal region, is the favorite place for ulcers. In ulcer of the duodenum it is not very common for hemorrhage to occur, although occult blood is frequently found (the stools were not examined for occult blood). Even though bleeding may occur it is very infrequent. However, there is a tendency in duodenal ulcer, as in gastric, to vomit. A great many duodenal ulcers are chronic and the attacks intermittent, differing somewhat from gastric attacks, and may be very severe. This woman's attacks do not coordinate with our true picture of duodenal ulcer, but they do come closer to the picture of duodenal than gastric. The emaciation may be due to spasm of the pyloric ring. The diagnosis, duodenal ulcer, holds first place in my choice of diagnoses in this case, and it is the diagnosis agreed upon by our group.

(Dr. McWhirt)

Our case is a woman 58 years of age, who comes to the out patient department for relief of pain in the stomach and gas. She brings a letter from her attending physician which gives the diagnosis of gastritis and neurasthenia. We hardly feel that this diagnosis will account for all the symptoms and findings in this case.

A patient who has lost, over a period of four years, sixty pounds, and in the last few months thirty-five pounds or more, makes us think very seriously of malignancy. We first considered malignancy in the region of the stomach, but the classical symptoms are absent; malignancy of the liver, but there is no acites or edema; malignancy of the pancreas,—marked jaundice and cachexia are present in this disease,—they are absent in our case. Ulcer of the stomach: while this possibly is the cause of our trouble, our group feels more inclined to duodenal ulcer. We have to rule gall bladder out of the picture as there apparently is a negative Graham test. Ulcer of the duodenum will account for these occasional attacks of indigestion with the periods of well-being in between. In duodenal ulcer of long standing we always have a chronic duodenitis and with this condition, inflammation of the bile and pancreatic ducts with occasionally a low grade pancreatitis. In this condition there is occasionally a slight enlargement of the spleen. If our deduction of possible low grade pancreatitis is correct, this would account for a considerable part of the loss of weight, considering that she has pyorrhea, chronic tonsillitis, together with endocarditis.

The diagnosis of our group is duodenal ulcer, duodenitis chronic, inflammation of the gall ducts, inflammation of the pancreatic duct and sub-acute endocarditis.

Discussion by Massachusetts General Hospital Staff

(B. THURBER GUILD, M. D.)

I feel that gastritis is a very poor diagnosis to make unless it is toxic, as in alcohol or iodine ingestion. Neurasthenia is a very difficult diagnosis to make and I prefer to leave it for others!

"She dated the trouble from a hysterectomy for fibroids of the uterus thirteen years previously." That statement conflicts somewhat with the fact she came in complaining of disturbance of two months' duration. "She was in good health until three years before the visit" is another discrepancy. I get the impression from the history that she probably had disturbance of digestion for some period of time, and tha tthese dates were fixed in her mind when the condition was more acute.

I saw this patient in the Out-Patient Department, and I remember that this pain was a very definite thing with her, also that it was definitely relieved by eating a small amount of food or by soda.

In considering the question whether this might be gall-bladder disease I do not think that we can feel as a point in favor that the Russian oil gave the relief that an animal fat might have given by emptying the gall-bladder. I do not think a mineral oil would act in that way. It might however have coated over an ulcer of the stomach and thus relieved.

The statement about bowel movements is rather against cholecystitis.

Ten days before the visit is, I believe, the first time that she had jaundice. We could get nothing further back than that.

The loss of weight of course, turns our attention to the question of malignancy. It could have been explained by the diet she might have been following for peptic ulcer, but is not in line with the dianosis of cholecystitis.

"Occasional diarrhea" makes us think of malignancy and possibly gall-bladder. Dark urine could mean bile. Pyrosis would be less favorable for a diagnosis of malignancy and more suggestive of ulcer.

I think in the face of later data we shall have to believe that "heart not enlarged" was an erroneous finding. The heart would have to be enlarged with that pressure. Probably the aortic sound was transmitted to the apex rather than an apical sound to the aortic region.

The mass could be any one of the three things we are considering.

She was put on a five-meal bland diet—the idea being that we were dealing with a duodenal ulcer. It might not be duodenal because of the early pain after eating.

We were still considering the possibility of malignancy and cholelithiasis with cholecystitis but ulcer symptoms seemed to predominate. I think the x-rays were reported as negative. Sluggish peristalsis seemed a peculiar finding in view of the fact that she had pain so soon after eating.

Patients frequently think they are jaundiced when they are not; but if they are presumably jaundiced and have itching it is a little more determined.

Whether the reaction in her blood pressure was due to having her teeth extracted or to the loss of weight I do not know.

We considered chronic pancreatitis but ruled it out; also possible malignancy of the pancreas secondary to a gall-bladder condition, but she had no fatty stools and no glycosuria.

By a fortunate coincidence a student who was working with me visited one of our large hospitals and in walking the wards accidentally found our patient. The visiting man spoke of the case, made his diagnosis, and told what had been done. He rather laughingly remarked that she had been to the Massachustetts General Hospital, where we had been treating her for gastric ulcer. She had had cholelithiasis, was operated on, and was very much better. When our student told him how classical the symptoms were for ulcer he was much more interested.

HOSPITAL REPORT

A report from this hospital is in brief as follows:

"January 3. This is an elderly, emaciated, jaundiced little woman who enters the hospital because of recurrent attacks of epigastric pain of three years' duration.

"There is a history of many years of attacks of upper abdominal indigestion and discomfort which are consistent with gall-stones and not inconsistent with an ulcer. There is a recent history of more severe acute attacks and of jaundice beginning about two months ago. She has been under observation for some time at the Massachusetts General Hospital and we understand that ulcer was tentatively diagnosed but that gastro-intestinal studies were made which failed to show it. They were about to pursue investigation of the gall-bladder further when the present acute attack caused her local physician to advise her immediate admission to this hospital.

"**Local examination** shows a palpable liver border, practically no tenderness over the gall-bladder region and no gall-bladder to be felt. The spleen is distinctly enlarged, the edge coming just below the costal border on deep inspiration. Patient shows a marked icteric tint of the skin and sclerae. Bleeding and clotting time are normal. It seems beyond doubt that she has just recovered from a very severe attack of gallstone colic with the passage of a stone into the common duct. Whether it is still there or not cannot be told. In spite of the jaundice there is plenty of bile in the stools. No noteworthy elevation of leukocytes so that no cholangitis seems to be complicating the situation.

"**Operative note,** January 7. Cholecystectomy. Choledochostomy for chronic cholecystitis and cholelithiasis. Excision of small myoma of stomach. Hepatic cirrhosis."

"**Roentgen study,** January 30. The esophagus appeared normal. The stomach showed good position and tone, active peristalsis, a smooth outline and no six-hour residue. The duodenal cap showed a deep notch on the lateral or greater curvature border, but was not demonstrably fixed or tender. The ileum was normal. The cecum was tender but well filled and freely movable. Motility was good. Impression: Duodenal ulcer.

"**Discharge note,** February 19. The patient is discharged today to return to the Gastro-Intestinal Clinic in three weeks, where her duodenal ulcer will be treated. In the meantime she has been instructed to take a five-meal Sippy diet with powders p.c. Inasmuch as it is is causing her no symptoms at the present time, and also because of the fact that the ulcer appeared to be a chronic, perhaps 'healed' one **when seen at the time of operation,** it is very probable that the gall-bladder was the etiological agent in producing the patient's symptoms prior to the operation, inasmuch as the pain was typically gall-bladder in type.

"**Diagnosis:** Cholecystitis, chronic. Cholelithiasis. Fibromyomata of stomach wall. Hypertension. Duodenal ulcer."

FURTHER DISCUSSION

My impression is that if she had jaundice the bleeding and clotting times would not be normal, but I am not sure about that. On the other hand if she were having brown stools at that time and no bile in the urine, her blood may have become normal though she still showed a jaundiced skin.

What treatment she had in the interim between the operation and her discharge I do not know; probably some form of Sippy diet.

I chose to report the case because I do not remember finding another case with both ulcer and gall-stones in the same patient, but I am inclined

to think that infrequently they may be present together. Some investigators talk about gastric ulcer being the result of infection, and if there is infection in the gall-bladder it may produce duodenitis. She certainly had classical signs of ulcer, also as classical as they can be of cholelithiasis, and according to the operation and later x-rays she had both conditions.

DR. CABOT: Did they see anything of the ulcer at the time of operation?

DR. GUILD: The report states that the ulcer appeared at the time of operation to be a "chronic, perhaps healed" one. The loss of weight seems to me excessive for that condition or the combined condition. Possibly we may have another diagnosis to make in the future.

DR. CABOT: They found no ascites?

DR. GUILD: None in the report.

DR. CABOT: What can you tell us about this, Dr. Holmes?

DR. GEORGE W. HOLMES: As these notes read, the Graham test showed no visible stone. Is there a possibility the dye was not given?

MISS PAINTER: They asked for a Graham test.

DR. HOLMES: There may have been another examination. This is a plain film of the gall-bladder and fails to show any stone. Of course, if the patient had had the dye, it would mean that she had a diseased gall-bladder. If she had not had it, it would mean practically nothing. Was that appointment for a re-examination of the stomach or of the gall-bladder?

DR. GUILD: They asked for a Graham test on the record.

DR. HOLMES: Sluggish peristalsis or hyperperistalsis means very little unless we have other things to go with them. The same patient may show sluggish one hour and hyperperistalsis the next. The first time the patient is often frightened and the stomach does not move at all. She had had one x-ray examination when Dr. S. saw her, and had probably got over her fright. We did mention that the duodenum was irritable. That of course means that they had difficulty in filling it and were not quite sure whether it was normal or not. What we usually do in these cases is to suggest that the examination be repeated. If we did not do it I should say we were open to some criticism for not going so.

MISS PAINTER: She entered the other hospital, and did not come back here.

DR. CABOT: There is a phrase in the other hospital record which I wanted you to interpret: "The duodenal cap showed a deep notch in the lateral or greater curvature border." What about that notch? What does that mean?

DR. HOMES: It probably means an incisura opposite the ulcer. Such a notch may be due to spasm from any cause. That alone would not be sufficient, I think, to make the diagnosis. But perhaps it means more than that to the man who uses that term. Some notches would be significant, others would not.

A PHYSICIAN: The patient in that particular instance was having gall colic pains. Would that give a notch?

DR. HOLMES: The duodenal bulb is apt to be deformed in gall-bladder disease and may be misleading. We are beginning to think that when we get a persistent deformity of the cap it usually means ulcer. This appearance of spasm and all those things are becoming of less and less value. We are studying the cap and finding ulcer earlier and smaller, and the number of cases in which the vomiting can be attributed to some other cause is getting less and less.

DR. GUILD: I would like to know if in the necropsy findings you have found this combination?

DR. TRACY B. MALLORY: I think we do not very often find marked degrees of the two together. A good many patients with gastric ulcer show mild chronic changes in the gall-bladder, just as almost every appendix shows remnants of infectious changes. We very seldom find a perfectly normal gall-bladder. From one point of view we could say that the gall-bladder is frequently diseased in patients with gastric ulcer, but I believe it is not to a serious degree.

DR. CABOT: Is it any more diseased in peptic ulcer than it is in other conditions?

DR. MALLORY: I think not.

DR. CABOT: Just the "normal amount" of gall-bladder disease.

DIAGNOSIS

Duodenal ulcer.
Chronic cholecystitis.
Cholelithiasis.

EPIGASTRIC PAIN, VOMITING AND DELIRIUM

(Cabot's Case No. 14101, New England Jour. of Med., Apr. 26, 1928 p. 518.)

Below will be found the discussions of selected teams from the Maricopa County Medical Society, at their meeting of October 15th; of the Yavapai County Medical Society and Medical Officers of U. S. Veterans Hospital at Whipple, Ariz., at their meeting of October 16th; and diagnoses from the Yuma County, Ariz., Medical Society.

For the Case History, see September issue of this journal, p. 413.

Discussions by the Maricopa County Medical Society.

DR. GEORGE C. TRUMAN, Mesa, Ariz.
DR. R. J. STROUD, Tempe, Ariz.

(Dr. Truman)

The case under discussion does not present a clear cut picture of any single pathological condition, but gives such a varied history and physical findings as to lead one to believe that several lesions may be present.

I do not believe that there will be any dissension from the opinion that we are dealing with an acute abdominal condition and that the autopsy findings will be mostly in the abdomen proper and not in the pelvis. The mode of onset was sudden, forty-eight hours before admission, with pain in the epigastrium and vomiting of blood. Twenty months before, menses had stopped, nor were they replaced by vicarious bleeding from any mucous membrane or it would certainly have been mentioned in the history, so we need not consider this a part of the menstrual cycle.

Upon physical examination there were found as positive signs of pathology: a small draining sinus in an old appendiceal scar, a tympanitic abdomen with no dullness in the flanks, pain on deep pressure in the right

hypochondrium and epigastrium, with fever. The appendix had been removed, so that it is not necessary to consider that as a factor in the condition present. A sinus from the old appendectomy was present and from its chronicity one is led to believe that the original appendiceal involvement may have been tuberculous in character. However, the peritoneal irritation present is more than likely not tuberculosis because it is of short duration (forty-eight hours), and is not accompanied by any fluid. No mention is made of jaundice, and bile was found in the stool, so that there was no stoppage of liver function nor interference with the passage of bile from the liver to the duodenum.

In reviewing the history of this case there is only one thing that stands out. The acute illness started with the vomiting of a quanity of blood. Where did the blood come from? The diagnosis of the case probably hinges on this point alone, since all of the acute symptoms followed after this vomiting. Vomiting blood may come from the stomach or other parts of the gastro-intestinal tract. Bleeding from the stomach may be from local or general causes. The local causes are again divided into direct and indirect.

The direct local causes include ulcers, simple and tuberculous, diphtheritic and septic; gastritis, simple, acute and chronic; varices and miliary aneurisms; cancer and trauma. The trauma may be physical and chemical. Among the physical causes are foreign bodies, the strain of vomiting, injury from the stomach tube and acute distention. Among the chemical causes are poison, emetics and purgatives.

The indirect local causes include portal obstruction, cirrhosis of the liver, syphilis of the liver, atrophy of the liver, pressure on the portal vein, portal thrombosis, thoracic disease with circulatory obstruction, emphysema, chronic pleurisy, fibroid lung and organic heart disease. In bleeding due to general causes, there are hemorrhages from other mucous membranes besides the gastric. The general causes include sepsis, the exanthemata, typhoid, hematemesis neonatorum, autotoxic states, anemia, blood dyscrasias and diseases, hemophilia, purpura, scurvy, pernicious anemia, leukemia, neuropathies, epilepsy, tabes, general paralysis, hysteria, meningitis and vicarious menstruation.

Bleeding from epigastric sources may be due to esophageal varices, abscess contiguous to the stomach, or ulcers or fistulas may open up a tract leading to the stomach resulting in hemorrhage. Aneurism of the abdominal aorta or celiac axis may perforate the stomach and cause hematemesis.

Taking up the direct local causes of bleeding, ulcers of the stomach or duodenum cannot be eliminated; in fact the past history would lead one to suspect that an ulcer may be present. The fact that the vomiting was not continuous would seem to eliminate any form of gastritis. There is no history of trauma or the taking of poison, so that physical and chemical agents need not be considered. There was no cachexia present which would seem to speak against cancer.

Considering the indirect local causes we have cirrhosis of the liver. There was no jaundice present, neither was there any ascites. The x-ray did not show any marked evidence of thoracic disease, and physical findings showed only a murmur at the apex which was not transmitted. These murmurs are sometimes found following hemorrhage, so we cannot say that the hemorrhage was due to it.

In considering the general causes of hemorrhage, we can eliminate at once all of the blood diseases such as pernicious anemia, leukemia and hemophilia, as the blood picture showed only a secondary anemia. There is no history of epilepsy. There is a possibility however that there may be an infarction of some of the abdominal arteries. The usual one is the mesenteric. The source of the infarction could be the chronic infection in the sinus lying in the appendectomy scar. The Wassermann is negative, so also is the history for luetic infection. Vicarious menstruation has already been eliminated from consideration.

We will give the extra-gastric causes of bleeding little consideration because the hemorrhage was not excessive nor was it accompanied by any visable amount of pus such as would happer if an abscess had ruptured into the stomach.

By process of elimination we have arrived at two possible causes of the hematemesis in the case. They are gastric ulcer and thrombosis of arterial circulation especially the mesenteric artery.

The rapid pulse, high temperature and distention speak for two other possibilities; that is general peritonitis such as would follow a ruptured gastric ulcer and acute pancreatitis which can also be accompanied by gastric hemorrhage as one of the general causes.

The diagnosis lends itself to four possibilities:

I and II. Ruptured gastric ulcer with peritonitis.

III. Thrombosis of the mesenteric artery.

IV. Acute pancreatitis.

The pain and collapse was not severe enough for acute pancreatitis, which leaves the first two possibilities. My first choice would be ruptured gastric ulcer because of the previous history of gastric distress with mesenteric thrombosis as second choice.

(Dr. Stroud)

This case has a lot of features that are interesting, and yet irrelevant to a diagnosis. I shall try to analyze a few of the points which go to make up the case, as Dr. Truman has taken it up from a standpoint of the significance of bleeding from the stomach, and the possible diagnoses with the final diagnosis, as far as can be determined from the meager history.

This young woman has had an irregular catamenia, probably the result of thyroidectomy. The uterus has lost its function because of endocrine deficiency, by the loss to the ovaries of the thyroid stimulation, just at the time of life when the stimulation was most necessary. Then for twenty months the necessary hormones were entirely lost and no menstruation has taken place. We do not know just how much this had to do with her despondency, but the suicidal intent could come from this lack of ovarian stimulation. In fact, it is with some trepidation that surgeons remove ovarian tissue from young women before thirty years of age. The menses stopping, there were certain symptoms referable to the stomach at the time when they should have commenced, and continuing one day. There is no mention that blood was in the stools as this may not have been macroscopic or noticed if able to be seen. How much of a vicarious menstruation is here, we do not know. Then when the physical examination was done, no mention of infantile uterus or other abnormal condition of the adnexa is made. On the other hand, the lack of apparent vitality can be explained by the fact that she must have had somewhere, at some time, some debilitating disease with either an absorption or toxemia, or a loss of blood from some cause. The case history says that two years before admission, she had an appendicostomy for colitis, and then states further that she had a mucous colitis of twenty months standing, an apparent error in history taking, for the original colitis must have been a mucous colitis, and was no doubt of some time standing before the operation.

Taking up the question of mucous colitis and causes, we have first, amebic dysentery, acute or chronic. She gives no history of an acute attack of dysentery, and no data is given us as to work done on stools. Mucous colitis can also be caused by coli or streptococcus organisms lying in the folds of the colon, and propagating there, or the streptococci or other organisms may have their habitat in the gall bladder, appendix or the edges of a stomach ulcer, and quantities of them may be propagated at various times to set up a mucous colitis. Then the pressure from growths, such as tumors of the adnexa, retroverted uterus etc., may set up a mechanical colitis, or at least destroy the resistance of patches of the colon to organisms.

We note also, a blood pressure of 130-70, and pulses are described as not of good volume and tension. This statement of pulses not being good volume, is challenged by some clinics and yet there is a clinical distinction between a good and a bad pulse. The old drainage of the appendix probably has nothing to do with the picture. The soft blowing murmur (systolic) of the heart would be heard in any heart embarrassed as this was. The blood shows an active fight against some organism, and that a massive battle between the organism and the host is going on, with the host on the losing end, because of the anemia as shown by the blood counts, and pale mucous membranes.

The x-ray picture of right lung would rather point somewhat indefinitely to pressure upwards from the liver, or below the diaphragm, for it is less radiant than the left, especially at the base. The urine on the day of admission was negative. Two days later the kidneys showed signs of embarrassment, and that some acute process had affected it. There is no doubt of some cloudy swelling, and the presence of pus would indicate an active lesion. The abdomen being distended and tympanitic throughout, with tenderness over the right upper quadrant and epigastrium on deep palpation, rather points to an entity in that region. Still there are no outstanding and definite symptoms of any kind which lead one to a definite diagnosis of trouble below the diaphragm. Dr. Truman has already gone over the bleeding from the stomach. It is possible in all cases that idiopathic bleeding may take place from the stomach, especially when in single amounts. The remote abdominal conditions that could give such a picture, would be towards the mesentery or omentum, besides the conditions mentioned by Dr. Truman.

The lumbar puncture shows a spinal fluid count of 350 leukocytes. This, no doubt, is just part of the picture of the terminal

infection, and does not get at the original cause of the trouble. Acute meningitis would give this picture, although I believe it is terminal to the original cause. Pus in the kidney, and this finding in the spinal fluid, seem to me to be a terminal thing with the original cause elsewhere. Blood culture is not mentioned.

My diagnosis, is therefore, that of some debilitating disease below the diaphragm with an acute attack developing suddenly which gave rise to bleeding from the stomach, and further to peritonitis with some mass high on the right side below the diaphragm, and with a spreading of this infection throughout the body to give a terminal meningitic and kidney condition. While indefinite, the closest diagnosis would be a chronic ulcer of the stomach on the posterior surface, rupturing into the lesser peritoneal cavity, producing tenderness and a mass, and which went on to peritonitis, and terminally the organisms finding their way through the body, to distant organs, as the kidney and brain. I know of no other entity that will quite fit the conditions, although the fit is imperfect.

Discussions by Yavapai County Medical Society and Medical Officers of Fort Whipple, Group. I.

DR. J. T. MALONE, Whipple, Ariz.
DR. JAMES H. ALLEN, Prescott, Ariz.
DR. C. C. BENEDICT, Whipple, Ariz.

(Mr. Malone)

One of the most significant factors in the history of this case is the statement that forty-eight hours before admission the patient expectorated a cupful of brownish-red blood. Going a little deeper into her past history we find that thirteen years before her admission she had a thyroidectomy performed. We are rather surprised that a girl of twelve years should have been submitted to such radical surgical procedure, as we rather suspect the usual type of thyroid involvement occurring at that period. It appears, however, that some endocrine unbalance has persisted since the removal of the thyroid, for we are told that her menses have always been irregular, that she has been depressed, melancholic, and had attempted suicide. Moreover, the history tells us that twenty months prior to admission to the hospital she had a complete suppression of menstruation, and that coincident with the cycle she had a moderately severe epigastric pain. You will recall that at the onset of this present illness, and associated with the blood spitting, she had epigastric pain. We have considered the possibility of vicarious menstruation to account for the hemoptysis. We realized the necessary requisites in making such a diagnosis and ignored it in the belief that we shall be able to convince you that this bleeding did not originate from a normal organ. Ectopic pregnancy was likewise considered as a possible factor in the differential diagnosis and will be further discussed by my colleague. Examination revealed a fairly well developed and nourished woman. On examination of the chest we are told that a systolic murmur was noted at the apex. We were more or less interested in that murmur in our preliminary perusal of the history, for we thought of a vegetative mitral valve and a resulting brain embolus. We are told that the heart was not enlarged and the murmur was not transmitted. Furthermore a roentgenogram of the chest, taken perhaps as a routine, did not mention a suspected cardiac enlargement. Therefore, it appears that we are justified in ruling out a cardiac valvular involvement, consider the murmur as a functional one, and dismiss the heart from the picture. We are however, deeply interested in that roentgenogram of the chest, not from the standpoint of suspected pulmonary pathology, but rather as a suggestive pneumoperitoneum under the right diaphragm. It is true that the roentgenologist merely stated that the right lung was less radiant than the left, especially at the base, and with this exception the lungs were normal. We are interested in the opacity at that base for the reason stated above and the linking up of the picture with a perforated duodenal or gastric ulcer. In a recent article by Gibson of New York in the Journal A.M.A., (reporting a series of perforated gastric and duodenal ulcers), air in the region of the right diaphragm was a predominating factor in the differential diagnosis of acute abdominal conditions. The abdomen was somewhat distended, tympanitic and tender over the right upper quadrant and epigastrium. We do not have a typical picture of a severe peritonitis nor would we suspect same in the time interval. Gastric and duodenal contents are not highly septic, if at all. But we believe we have a picture of an early peritonitis associated with a perforated peptic ulcer. The history further relates that she had had an appendicostomy two years before admission, and that at the present time there is a fistula over the site of the operation scar with a slight purulent discharge. Her stools were of the colitis type and examination revealed gross blood and bile. We presume that a diagnosis of amebic dysentery had been previously made and the appendicostomy

was for the purpose of irrigating the bowel. They tell us nothing more, however, in the history that would cause us to presume otherwise. The connection of this existing colitis with the terminal picture will be considered later in the discussion. The blood and urine chemistry give us very little information except perhaps to denote a passive nephritis, not primarily in the picture, and a leukocytosis with suggestive secondary anemia. The spinal fluid was perhaps the most interesting finding in the entire history. We made an exhaustive search of the literature in an endeavor to find what type of brain pathology would present such a picture of discrepancy. I refer to the reported findings that the spinal fluid showed 350 leukocytes with normal dynamics and normal chemistry. We must, however, presume that no error was made and that a meningitis existed. You will recall I mentioned that we were interested in the possibility of an embolus from a damaged heart valve but later ruled this out. We are now concerned with a possible septic embolus in the brain to account for the spinal fluid findings and we believe we can demonstrate the source of the embolus in the abdomen, probably from a mesenteric vessel, associated with either a ruptured duodenal or gastric ulcer, or an acute hemorrhagic pancreatitis. The possibility of these two conditions co-existing, or existing separately, will be the basis for the differential diagnosis to be continued by my colleagues.

(Dr. Allen)

Dr. Malone has stated the outstanding symptoms in this case: severe epigastric pain, vomiting of a cupful of brownish-red blood and the passage of blood and bile in the stools. In addition we have a very fulminating condition which caused marked prostration with death in four days from the inception of the illness. The temperature indicates infection. The blood picture may be due either to a septicemic process or it may be due to an acute blood loss from hemorrhage. Immediately following a hemorrhage, there is a polymorphonunclear leucocytosis, which may be as high as 40,000. My colleague, Dr. Malone, has considered conditions outside the digestive tract which might account for the symptoms presented here. We believe the epigastric pain, vomiting of blood and passage of blood in stools indicate that the cause of this condition is in the digestive tract. There is a history of colitis which could cause trouble by infecting other parts of the digestive apparatus. In addition, there is a draining fistula over the appendix which could also cause it. Among conditions which should be considered are pylephlebitis, cholecystitis, mesenteric embolism or thrombosis, ruptured gastric or duodenal ulcer and acute hemorrhagic pancreatitis. Cholecystitis could cause the epigastric pain but there should be some enlargement over gall bladder. There is no icterus or muddiness of the skin and the stools show the presence of bile. In addition there is no rigidity in right upper quadrant and the fulminating type of illness which we have here is not usually associated with gall bladder disease with so few localizing symptoms.

Pylephlibitis could account for the fever, leucocytosis and marked prostration but not for the bloody vomit and blood in stools and it is believed the pain would be generalized instead of being limited to the epigastrium.

A mesenteric embolism or thrombosis, especially one involving the superior mesenteric artery could cause the pain, bloody stools and prostration. However, in thrombosis and embolism the vomiting is usually only blood-stained, the temperature as a rule is subnormal and we have at first an obstinate constipation, later followed by a bloody diarrhea. In this case we have an elevated temperature and have no history either of constipation or diarrhea.

We now come to the last two conditions above mentioned—a ruptured gastric or duodenal ulcer and an acute hemorrhagic pancreatitis. We believe that either one of these conditions or both of them could account for the pain, generalized tenderness, extreme prostration, bloody vomit, bloody stools, the blood picture and elevated temperature. My colleague, Dr. Benedict, will discuss in detail these two conditions. Our diagnosis is:

1. Acute hemorrhagic pancreatitis.
2. Ruptured gastric or duodenal ulcer.

A general peritonitis complicated either one or both of these conditions.

(Dr. Benedict.)

Group I has unanimously agreed upon one thing—it is very hazardous to make a positive diagnosis in this case. It will converge upon acute hemorrhagic pancreatitis or ruptured gastric or duodenal ulcer, with a leaning in favor of pancreatitis. In order to save time, I have drawn upon the board a diagram of the anatomy of the region of the pancreas, and a classification of the causes of hemorrhagic pancreatitis, the first by Tice and the second by Speese. You will notice that Tice gives first place to enterogenous, and Speese to lymphatic. To quote E. O. Rosenow, "In infections of

bones, joints and endcardium, the infection travels through the blood; in other words, hematogenous. In infections of those organs that communicate with the surface of the body, such as the stomach, gall bladder, pancreas, etc., these infections come very often from the intestinal tract or ducts leading into it." And in connection with infections of the intestinal tract, the bacillus coli is not the usual offender in acute conditions but is common in such a chronic condition as our case had. The fact that the infection would have to travel some distance from the appendix or colon, or wherever this colitis supposedly was located, to reach the pancreas, rather argues against such a source of infection. In regard to the lymphatics, Sudler has done some very extensive work, tracing the lymphatics of the digestive tract, and in 1911, Frank, working independently, positively established the communication existing between the lymphatics supplying the gall bladder and pancreas. Also that it is not impossible for infection in the gall bladder to back up into the pancreas through the duct of Wirsung. The common bile duct empties into the duct of Wirsung before entering the duodenum, which entrance is usually by one opening. Acute infections by way of the lymphatics are very rare, but chronic infections are common. In regard to infection by way of the ducts, there is this to prevent it—the pancreatic secretion is bactericidal in power, and just at the point of emptying are folds of mucous membrane that mechanically protect the opening from infectious organisms. Archibald says he has produced acute hemorrhagic pancreatitis by introducing into the gall bladder of cats a culture of bile, or bile salts, incubated for various lengths of time with bacteria, and by forcing this to enter the pancreatic duct by spasm of the sphincter, owing to sudden rise of pressure in the gall bladder or to application of hydrochloric acid to the mucous membrane of the duodenum. Mann, in Mayo's Clinic, speaks of vomiting and retching as a means of spreading infection through the duct of Wirsung into the pancreas. He says vomiting and retching will raise intraduct pressure 1000 m.m. bile, which would account for some infections travelling up the duct and producing a pancreatitis. Regarding the existence of chronic cholecysittis with acute pancreatitis,—the common bile duct passes down behind the pancreas in a depression in its posterior surface, thus offering a good chance for spread of infection in case of infected bile. E. S. Judd reported 1200 cases operated for cholecystitis in which 26.8 per cent had pancreatitis. In regard to hematogenous source, it may occur in chronic but seldom in acute infections; in acute infections such as typhoid, influenza or mumps it occurs once in a while; but it does occur in furunculosis, abscesses and appendicitis, forming a part of the general systemic infection or septicemia. In perforating gastric or duodenal ulcer, the infected material is in close contact with the pancreas, thus affording an opportunity for infection. The symptoms point towards pancreatitis as a preference to perforating gastric or duodenal ulcer. Tice says the patient may vomit blood only once in hemorrhagic pancreatitis, as did our patient; blood may be discharged into the duodenum directly from the pancreas, and be regurgitated. Epigastric pain might be due to another condition. The acetonuria is peculiarly significant of pancreatitis. Prostration is present and always terminates in sudden death.

Our diagnosis—preference of acute hemorrhagic pancreatitis or perforating gastric or duodenal ulcer, favoring the former; cholecystitis not entering into the picture to any great extent; a colitis, and apparently a peritonitis.

Yuma County Medical Society (Ariz.) Diagnostic Conclusions. Meeting of Oct. 9th).

Dr. Ketcherside:- Amebic Dysentery.
Dr. Shields:- Acute Miliary Tuberculosis.
Dr. Cain:- Perforating Ulcer of Stomach.
Dr. Reese:-Bichloride of Mercury Poisoning.

Discussions at Massachusetts General Hospital

RICHARD C. CABOT, M.D.

It is rare to have thyroidectomy at twelve, but we have to take the statement as given.

The remark about appendicostomy and colitis is surprising. In the first place one does not expect to see a person who has colitis so severe that appendicostomy is done recover as this person seems to have recovered. In the second place it is spoken of later as mucous colitis. This makes me suspect that is what it was in the beginning. If so, appendicostomy was the worst thing they could have done.

There ought to be more detail on the spinal fluid. We do not often get normal chemical contents with 350 leukocytes. I should think that fact of the leukocytes the most important fact that we have.

The complaint with which she came, abdominal pain and vomiting of reddish brown fluid supposed to be blood, does not easily associate itself with the final symptoms, especially with the lumbar puncture fluid. Knowing that she had had an appendicostomy we wonder if that and the adhesions might possibly have some connection with the abdominal symptoms; but I do not think the evidence is good. An appendicostomy ought to leave very little in the way of adhesions. In the second place her symptoms do not seem to be par-

ticularly in that region. There is nothing said about abdominal spasm. The abdomen is distended, tympanitic and tender. She has nervous symptoms, semi-delirium and coma. I should say the best explanation is general infection in which the nervous system took part and which showed itself with digestive symptoms in the beginning, rather than to say that local abdominal disease was the main cause of her death. Of course it is perfectly possible that she had a general peritonitis and that the meningeal irritation or meningitis is secondary to that.

The count of red cells looks as if this illness had been going on more than forty-eight hours, for it does not seem probable that she had vomited enough blood to cause that anemia, and there is nothing else to explain it. But what illness she may have had for more than forty-eight hours I have no idea. We have no evidence, I should say, of any chronic nephritis, although we do not know the gravity of the urine.

DR. RICHARD B. KING: It was perfectly normal.

DR. W. PEARCE COUES: Do you think she could have taken some poison a few days before admission to the hospital in a suicidal attempt?

DR. CABOT: That would have caused her vomiting, but I do not see how we can easily link that up with the condition of the lumbar fluid or with the anemia. I do not know any poison that will cause such an anemia in two days. There is not any evidence of jaundice or anything like acute liver trouble. I do not see how we can say she has tuberculosis in any form or syphilis in any form.

A PHYSICIAN: Can it be endocarditis?

DR. CABOT: The history must be very defective if that is true.

A PHYSICIAN: She has a systolic murmur.

DR. CABOT: Apparently she was sick only forty-eight hours. If she had had endocarditis I should have been satisfied to explain the abdominal symptoms in that way; but I should say that we have no good basis for a diagnosis of endocarditis in any form.

A PHYSICIAN: How do you explain the bloody vomits and bloody stool?

DR. CABOT: We see a great many critically ill patients, especially on the surgical side, vomit reddish brown fluid. This happens in peritonitis often, but also in a great many cases in which we never find a cause. The stool examination showed gross blood, but we do not know as to the question of hemorrhoids. We have no rectal examination. She is said to have had a colitis, not however of the type that ordinarily shows gross blood. Possibly her anemia is due to old colitis. If she has lived here that colitis was probably not of the type likely to spread beyond the intestine. I do not believe that I can make a diagnosis.

DR. KING: She had a perfectly normal blood sugar and her CO_2 tension was about 40 volumes per cent. I saw her about twelve hours after she became comatose and thought she might be in diabetic coma because of the tremendous hyperpnea which she was said to have had for some hours.

DR. CABOT: You do not know any more about that leukocyte count in the spinal fluid, do you?

DR. KING: No sir.

DR. CABOT: That is the most definite fact we have. If only we had a little bit more!

DR. TRACY B. MALLORY: There must have been a high percentage of polymorphonuclears.

DR. CABOT: What makes you think that?

DR. MALLORY: From our examination of the spinal fluid post mortem.

DR. CABOT: Let us say then that they are polymorphonuclears. That means acute meningitis. Is that all there is? No, because acute meningitis does not cause anemia, and she has it. I think we must say some form of acute meningitis. Beyond that my only guess is that there is also some form of sepsis, local or general.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Acute miliary tuberculosis.
Tuberculosis peritonitis.

DR. RICHARD CABOT'S DIAGNOSIS.

Acute meningitis.
Sepsis, local or general.

ANATOMIC DIAGNOSES

1. *Primary fatal lesions.*
   Septicemia, staphylococcus aureus.
   Staphylococcus meningitis.
   Miliary abscesses in heart, spleen and kidney.
2. *Secondary or terminal lesions.*
   Chronic colitis.
   Focal necrosis of the adrenals.

DR. MALLORY: The primary diagnosis was septicemia with staphylococcus aureus. It showed up in the blood culture. Numerous miliary abscesses were found in the spleen, heart and kidneys. The lungs contained an old infarct which must have been due to a much older process than this. No source or suggestion as to where it might have come from was found. The brain showed a definite purulent meningitis also due to the staphylococcus aureus. She did have a true chronic colitis without any gross ulcers but with very marked thickening of the wall of the sigmoid colon and a typical cellular infiltration which extended down through the mucosa and muscularis into the serosa. No source for the sepsis was found. Whether it was possibly an extension from the colitis, I cannot say. It hardly seems likely.

DR. CABOT: Is the organism of this form of colitis known, in your opinion?

DR. MALLORY: Not certainly. From a very considerable proportion of them we can recover streptococci, usually of the viridans type. A special diplococcus has been found in many cases by Barben of the Mayo clinic and he has also had very favorable results from specific vaccine therapy. I do not feel, however, that the etiology can be considered settled.

DR. CABOT: Do you take it the colitis is the cause of the anemia?

DR. MALLORY: The sepsis may have persisted very much longer than the history suggests. The kidney showed not only acute miliary abscesses situated around infectious thrombi in small blood vessels, but all stages of healing abscesses and all stages of septic infarcts. So that I think the process must have lasted several weeks without much question.

A QUESTION OF UREMIA

(Cabot's Case No. 14121. New Eng. Jour. of Med. May 17, 1928, p. 693).

Below will be found the discussions of selected teams from the Yavapai County Medical Society and Medical Officers of U. S. Veterans' Hospital at Whipple, Ariz., at their meeting on October 16th; discussions of the Maricopa County Medical Society at their meeting of October 15th.

For the case history see the September issue of this journal, page 414.

Discussions by Yavapai County Medical Society and Medical Officers of Fort Whipple, Group III.

DR. I. D. LOEWY, Whipple, Ariz.
DR. B. L. JONES, Whipple, Ariz.
DR. E. A. GATTERDAM, Whipple, Ariz.

(Dr. Loewy)

This case presents the characteristic history of an emergency case; the patient lived only five or six days. There is a sparsity of history and practically nothing but objective symptoms. The laboratory findings are rather scanty. Our attention is invited to the kidneys, heart and brain. The preponderance of symptoms found upon examination points to some condition in the brain. The kidneys are involved as shown in the urine examination—a trace of albumin, rare hyaline casts and much pus; it does not tell whether it was a catheterized specimen, or where the pus came from, whether from urethra, bladder, ureter or kidney. There was evidently a low grade nephritis, which may not have any bearing on the diagnosis. The heart is dilated, transverse diameter widened, with mitral regurgitation, a systolic murmur transmitted to axilla, which perhaps would give us a diagnosis of endocarditis. The most prominent symptoms are referable to the nervous system and especially referable to the right side of the brain. We have transient convergence, which shows irritation of the base, smoothing out of left side, ptosis of left eyelid; all point to lesion in the right side of brain. Left knee jerk absent and right impaired. In a later examination she does not use right corner of mouth, which suggests left side instead of right, but we believe the major condition affects the right side. A study of the spinal fluid was made but very meager details are given, the chemistry is entirely missing; the fluid was under considerable pressure, cloudy and of pinkish colour; microscopic examination showed red cells, rare mononuclears and very rare polynuclears. We looked up all authorities on spinal fluid and the presence of red cells in spinal fluid was not considered characteristic, a great deal more stress being laid on the chemistry and white cells, but we believe there was not sufficient number to be considered pathological. Something increased the endocranial tension to the point that it shows effect on the spinal fluid. In addition to that the history states on two examinations, the right eyeball was more prominent than the other; there is increased endocranial tension encroaching upon the orbit, pushing the eyeball forward; there is no tumor mass mentioned to account for the prominence of the right eyeball.

The differential diagnosis will be taken up by Dr. Jones.

(Dr. Jones).

In considering this case we must look at the question of the nervous system and the question of the cardio-vascular system, including the kidneys. There is a scarcity of laboratory findings. Taking up first the question of spinal fluid in this case, the history states that the fluid was pinkish, had increased number of red cells and that it was under pressure. Considering the case from the spinal fluid standpoint, we depend entirely upon the relation of that fluid to the physical findings, and in this case we must take up the question of brain tumor, brain abscess, meningitis, hemorragic encephalitis, apoplexy, which includes thrombosis, hemorrhage following ruptured embolism of the basal arteries, embolism which may come from this heart condition, and further a degeneration of arteries following a heart that shows degeneration. Considering first brain tumor, we do not think the physicians and surgeons in this case would have attempted to do a spinal puncture if they had the least idea of brain tumor, because of the danger of pressing down of the medulla, squeezing parts of the cerebellum and causing sudden death; further, this condition came on quite suddenly. We expect papillitis, projectile vomiting and severe headaches in brain tumor, and more neurological symptoms than we have in this case. Next considering brain abscess—a negative fluid would not necessarily help, it would depend upon whether that abscess had broken through and gotten into the ventricles, when there might possibly have been pus in the spinal fluid. Meningitis—we believe there was some meningeal irritation, but the spinal fluid was negative for organisms, negative for lymphocytes, for leukocytes and for increased amount of globulin; the lungs were negative, which would rule out tuberculous meningitis. We believe there was some brain irritation, but do not believe it was due to tumor, abscess, meningitis or hemorrhagic encephalitis. There was no marked fever, and the pulse was at times increased and at others slower. Blood in the spinal fluid—in cases of fracture of the base of skull, if it involves the arachnoid, it is fairly common. One method of determining the duration of this blood is by centrifugalization; if there is no hemolysis shown, it is attributed to recent hemorrhage and not to one of long duration. Cardiac condition—definitely enlarged heart, dilatation, arrhythmia; the

history states there is marked tension, and still give us a blood pressure of 120, which does not help much; this may be an extremis. There is no definite history to point to sub-acute bacterial endocarditis; it does say there has been cardiac damage as shown by the dilation, transmitted murmur to axilla and the arrhythmia; that may be an old endocarditis and not necessarily an acute condition. There is no history of rheumatic fever. There is a possibility of embolus broken from the heart valves, lodging in the brain, setting up inflammation and giving us these symptoms of paralysis of the face and eye symptoms, involving the third, fourth and sixth nerves. Along with the cardiac condition there are some renal symptoms; casts and albumin. We know there is, along with degeneration of the heart, degeneration of the arteries; there is the possibility of one of the arteries at the base of the brain becoming ruptured and the hemorrhage involving the third and fourth nerves and especially the nerve of the eye, giving us the strabismus, and possibly catching the facial nerves giving the paralysis of that side of the face. With regard to the absence of reflexes, this was definitely a terminal condition. We have eliminated tumor of the brain. We have a woman who has lost nine children; her husband showed no signs of syphilis; the eye symptoms, partial loss of light reflex, show a possibility of syphilitic contamination, but there is no positive Wassermann; she may have had syphilis and still die of some other condition, although this may cause hemorrhage and death.

Our differential diagnosis would be cardiac and renal disease with degeneration of the arteries, possible hemorrhage causing wet brain, which caused her death, hypertrophy and dilation of heart and mitral regurgitation.

(Dr. Gatterdam)

This case suggests infection. In handling this type of case in private practice an attempt at a diagnosis should always be made so that some sort of rational treatment may be given. The results of treatment or the autopsy findings will tell us if we were correct. Blood in the spinal fluid is interesting. It could have been caused by an injury to a blood vessel. Usually when we get small amounts of blood in the first fluid we say it is due to puncturing a blood vessel in doing the spinal puncture. Two other conditions that would cause it are skull fracture and a basal meningitis. Skull fracture should be thought of as we are apt to meet cases like this in emergency work. I had one case of a basal fulminating meningitis in which blood was obtained in the spinal fluid. This case was brought into the emergency hospital at 3 a. m. by a policeman. He complained of very severe headache. The policeman had found him on the street. The patient stated that his headache had started that afternoon and he had seen two physicians regarding it. One had told him he had a lumbago and the other said it was hysteria. On physical examination there was rigidity of the neck and positive Kernig; urine examination at this time showed a very large amount of albumin; blood count 23,000, and blood pressure 160/95; pulse full and fairly slow; spinal fluid contained much blood, so a culture or smear was not made. The patient lived eight hours after being admitted and autopsy showed an extensive basal epidemic meningitis.

In the case here discussed, physical findings point to three places. The heart, the kidneys, and the brain. From the heart examination it is quite probable that an endocarditis has existed. In other words there has been a previous heart injury, which predisposes to added infection or a subacute bacterial endocarditis, or endocarditis lenta. With this condition emboli occur which may cause infarction into the brain, kidneys, lungs, spleen, skin and other organs. The pus in the urine points to a pyelitis. This may be from infarction in the kidney from a cardiac embolism. Or this pyelitis may be primary and have caused an ulcerative endocarditis. Sudden death in this case points to a thrombosis or embolism of the coronary vessels. The drop in pulse from 120 to 60 may be due to a pulsus alternans and only every alternate beat counted, or due to cerebral pressure. It would also be accounted for by a partial heart block.

Turning to the neurological findings of partial paralysis one would suspect a cardiac embolism lodging in the brain. The paralysis seems to be first on the left side of the face and then on the right. It was not a true paralysis. In locating the lesion it appears to be in the vessels or tissues in the base of the brain. We do not look for emboli in this region but rather thrombosis of the greater veins, or sinuses. It is quite possible that thrombosis did occur, the infection originating from the heart. One physical sign here gives us the location, and that sign is bulging of the right eye. Headaches, partial paralysis, temperature, bulging of the right eye with no changes in the optic disc point to a thrombosis of the cavernous sinus on the right side. Our diagnosis is septicemia, subacute bacterial endo-

carditis, thrombosis of the right cavernous sinus, pyelitis.

Discussions by Maricopa County Medical Society, Phoenix, Ariz.

DR. F. C. JORDAN, Phoenix.
DR. M. E. WILSON, Phoenix.

(Dr. Jordan)

I will discuss the pathological conditions which we must exclude before making our diagnosis.

We have an Italian woman, age 42, brought to the hospital with a diagnosis of uremia. The diagnosis of uremia is quite frequently made with symptoms like these, and a great many times the diagnosis is wrong. Cabot says the diagnosis is always wrong where there is no past history of the ordinary symptoms of nephritis. The negative urinary findings and the low blood pressure are sufficient to rule out uremia.

Encephalitis must be considered. In this condition, the symptoms usually appear acutely, with a sudden rise of temperature, chills and headache. It may commence with convulsions, an apoplectic state, or progressive somnolence. The eye symptoms and palsies are not unlike those found in this case. Death may take place in the severe cases in a very short time. The spinal fluid contents in encaphalitis are usually characteristic; the fluid is clear, under slight increase of pressure, a very moderate number of cells which are nearly all lymphoctes. The symptoms of encephalitis are so varied that it can simulate almost any kind of brain lesion, yet we feel that the spinal fluid findings in this case would definitely exclude encephalitis.

In septic meningitis, a positive Kernig and rigid neck are practically always present. In all except the tuberculous form, the spinal fluid is cloudy and contains many cells with a large percentage of polymorphonuclear forms. The absence of these symptoms and the difference in the spinal fluid contents would be sufficient to exclude the different forms of meningitis.

In serous meningitis, which is usually secondary to some infectious disease, epilepsy or uremia, the spinal fluid is under pressure but the cell count is normal.

Trauma with subdural hemorrhage must be considered, since the spinal fluid findings may be identical with those found here. The absence of any known previous injury would be against this diagnosis, although there are authentic cases where the symptoms did not appear for a period of two years after the injury. Also the focal symptoms would be hard to explain on the basis of a hematoma caused by an outside injury.

The urinary findings were practically negative with the exception of large amounts of pus. This would indicate the presence of some inflammatory condition along the urinary tract. Cystitis, pyelitis, pyonephrosis and tuberculous kidney, are the most common causes of pus in the urine. We believe that this condition is entirely independent and has no relation to the immediate cause of death.

The blood and spinal fluid Wassermann, also the leucocyte count and blood chemistry would aid in the diagnosis. The finding of a bloody spinal fluid under tension in both punctures should rule out accidental hemorrhage caused by the needle.

We believe this blood in the spinal fluid came from the cranial cavity. My colleagues in this discussion will discuss the circulatory system and the brain.

(Dr. Wilson)

The men discussing this case are pretty well united in the opinion that the immediate cause of death was intracranial. The cardiovascular aspects of the case were studied mainly from the standpoint of etiology and in an effort to correlate the cardiovascular pathology with that suspected to exist in the brain or meninges.

The history points to one of two factors as causing the heart condition. She may have had a rheumatic heart or it may be affected by syphilis. The heart is very definitely enlarged in the transverse diameter: 11 c.m. to left of midsternum against a normal 7 or 8 c.m. There is a systolic murmur at the apex transmitted to the axilla, sounds of poor quality and an irregular pulse. These findings could indicate a rheumatic infection. The arrythmia could be due to auricular fibrillation or to impairment of the conduction system. The pulse rate is given in the inverse order 120-61. This may mean a varying rate due to arrhythmia or a decreasing rate due to intracranial pressure. There is no history of previous rheumatic symptoms, if we except the record of ten days fever two years previous to this illness. This could have been a tonsillar infection or a recurrence of a forgotten illness. We know that rheumatic symptoms may be light and the endocarditis following, nevertheless, severe. Given the possibility of an endocarditis, it could occur that upon the basis of an old simple endocarditis a purulent infectious process is engrafted (a case of this kind was recently reported to this society). As a possible origin for pus organisms, the history shows the urine to contain much pus indicating a focus in

the kidney or urinary passages. Infected emboli carried to the brain would explain the signs and symptoms if these were due to abscess or purulent meningitis. If some other origin for the infecting organisms could be found, it would be more reasonable to explain both the kidney and brain foci as being due to infected emboli from the heart, especially as the blood from the kidney goes through the lung capillaries before reaching the brain.

Another possibility that must be considered is that all the cardiac signs are due to syphilis. The history of nine dead children out of eleven stands out too prominently to be ignored. There is the suggestion of infection in the husband. If syphilis exists, it has been over a long period and without treatment. Cardiovascular and meningeal syphilis are symptoms of the later stages and come on five to twenty years after infection. The accentuated second aortic sound is quite characteristic and is usually sharp and metallic. The fact, that although the other sounds are poor and the blood pressure only 120, the accentuation exists, speaks for an aortitis. Arrythmia without symptoms of decompensation looks more like a lesion of the conduction fibers than auricular fibrillation. If the cardiac symptoms are luetic in origin it is probable that the brain lesion is also, the brain lesion being a meningeal syphilis, gumma, or an endarteritis.

The focal symptoms and nerve involvement point to the base of the brain and a broken down gumma or a ruptured small aneurismal dilatation would explain some of the symptoms, especially the blood in the spinal fluid.

The systolic pressure is only 120. Of course this may have previously been higher, and lower at present because of myocardial failure. A myocarditis would explain the mitral murmur if this is not caused by rheumatic endocarditis. A previous hypertension could be the cause of apoplexy; the few hyaline casts lends a little to this consideration, but on the whole, we do not think that the condition was due to apoplexy from hypertension.

A remote possibility, in view of the blood in the spinal fluid, is that myelogenous leukemia could be a factor. There is no corroborating evidence, no purpura, no enlarged spleen, no blood findings.

By process of exclusion, we narrowed the probabilities down to abscess, some syphilitic manifestation at the base—or something else. Dr. Robb will tell you which it is.

(Dr. Robb)

The patient was thought to be uremic. She had had headache and dimness of vision two weeks before entering the hospital. If this had been a case of uremia she would have had hemorrhages or signs of previous hemorrhages in the retina. The headaches could be caused by uremia, gastro-intestinal disturbances, ovarian diseases, apoplexy, brain tumor, brain abscess, syphilis, meningitis, chemical poisoning and many other things. There is nothing in the history to indicate that the headaches came from gastro-intestinal disturbances, ovarian disease, or chemical poisoning. Therefore it must be something inside the cranial cavity that caused it.

The loss of motion of the left side of the face, ptosis of the left upper lid and internal strabismus were, of course, caused by paresis of the seventh, third and sixth left cranial nerves.

Two years ago she was in bed with a fever. We might argue that she had a mastoiditis at that time causing pressure on the seventh cranial nerve as it passed through the fallopian canal, but there is no history of her having had any ear disease, such as suppurative otitis media. Besides, if the paresis of that nerve was caused by suppuration or pressure on the nerve as it passes through the mastoid bone, she would not have had paresis of the third and sixth cranial nerves. Therefore, the disturbance must be near the origin of the nerves.

Regarding the enlargement of the cervical glands; since the history does not state whether it was the superficial or deep chain and since there was no infection on the face or scalp we conclude that it must be the deep chain. This is of little help to us because we can have enlargement of the deep cervical glands from infections in the naso-pharynx, tonsils, meninges or brain substance.

We do not believe the headache and dimness of vision came from syphilis because if chronic syphilis had been present the eye grounds would have shown arteriosclerosis, probably chorio-retinitis and fine vitreous opacities. The history says that the fundi were normal.

As the headaches were of short duration and there is no history of vomiting and no choked disc we can eliminate brain tumors, with the exception of gliomata. Gliomata are such soft tumors that they can cause a great deal of destruction of the brain substance without causing pressure symptoms. The walls of the blood vessels in them are very thin and many times rupture, causing a hemorrhage at that place. We could in this way, account for the blood in the spinal fluid.

Since she had enlargement of the heart and possibly aortic disease, thrombosis of the longitudinal sinus might be thought of. This could explain the blood in the spinal fluid, for in such cases we have the brain bathed more or less with blood.

There are three causes for hemorrhage into the brain and spinal cord; trauma, extensive burns and diseased blood vessels. We have no history of trauma or burns. The blood in the spinal fluid could be caused by a prodromal or meningeal apoplexy and since she had no high blood pressure or arteriosclerosis in the fundi we do not believe this was apoplexy.

The low pulse of sixty-one, the temperature range, constant headache and stupor of the patient would suggest brain abscess.

Since she was very noisy, unreasonable and had rigidity of the neck, this would suggest meningitis in some degree.

Therefore we believe this woman had brain abscess near enough to the meninges to cause an erosion of the blood vessels which ruptured into the fourth ventricle and this caused pressure on the nuclei of the third, sixth and seventh cranial nerves and also on the direct pyramidal tract. This could explain the blood in the spinal fluid, the paresis of these nerves and the loss of the left knee jerk.

Discussion at Massachusetts General Hospital.

By RICHARD C. CABOT, M. D.

We should like to know whether her husband's pupils reacted to light or accommodation. We cannot say much otherwise.

The pulse tension was not high. In the old days we thought we were very skilful with our fingers, had the "tactus eruditus", the educated touch; but when we came to compare our guesses with the true measurements we found ourselves often very wrong.

We have not much evidence against that heart so far as I can see. We do not know that it is enlarged. We have the systolic murmur that we get in so many conditions without heart disease. I cannot say anything in particular on the basis of those facts.

They do not say anything about the muscular condition outside the face.

I do not see how we can say uremia on the basis of that urine.

40 cubic centimeters of spinal fluid is a great deal. 10 to 15 is what we get. usually.

DIFFERENTIAL DIAGNOSIS

This is a blind case. We have to say brain disease; but what disease? This is not the lumbar fluid of meningitis. If the report is right she has not meningitis. It is consistent with brain tumor or with cerebral hemorrhage. The normal fundi are strongly against tumor, that is strongly against death from tumor. If it is cerebral hemorrhage where is it? I do not know. The little that we have seems to point to the right side of the brain, but not strongly. "Hemorrhage" means some vascular lesion. We always ought to say that. Embolism, thrombosis or softening would give the same evidence. It is just as likely that this is softening as that it is hemorrhage. The presence of blood in the spinal fluid is the most troublesome thing. That seems more like hemorrhage than anything else. I should say cerebral hemorrhage, and I suppose it is on the right side of the brain. It might be in the ventricle or in the base.

A Student: What about lues?

Dr. Cabot: I think it is quite possible that lues is behind the picture. There is no conclusive evidence. Lues can go very well with the poorly reacting pupils. The condition of the knee-jerks in a person in her state does not mean anything.

A Student: What about cranial nerve paralysis?

Dr. Cabot. We have no good evidence of cranial nerve paralysis. It was suspected by one person. The mental symptoms are not characteristic. I do not know enough about the date of this case to know that it was not before the day of encephalitis. But I do not believe there was any encephalitis at that time. That is of course not quite a fair argument on the basis of the clinical facts.

A Student: What do you think of the increased pressure?

Dr. Cabot: We get increased pressure in cerebral hemorrhage or edema, sometimes in tumor, and sometimes with meningitis, especially "serous" meningitis.

Dr. Tracy B. Mallory: Did you make anything of the conjunctiva and convergence of the right eye, Dr. Cabot?

Dr. Cabot: I suppose I ought to, but I do not know what.

A Student: Nine children out of eleven are dead.

Dr. Cabot: That makes one think of syphilis, but it does not prove it. There are many other causes for the deaths of young children.

A Student: Are there not enough cells for meningitis?

Dr. Cabot: We ought to find more white cells.

CLINICAL DIAGNOSIS (From Hospital Record)

Cerebral syphilis?

Brain tumor?

DR. RICHARD C. CABOT'S DIAGNOSIS

Hemorrhage into the right side of the brain.

ANATOMIC DIAGNOSES

1. **Primary fatal lesions.**
   Thrombosis of the great venous sinuses of the cranium and of certain radicles of the superior longitudinal sinus and of the first portion of the right opthalmic vein and the first portions of the jugular veins.
2. **Secondary or terminal lesions.**
   Infarcts of the left lung.
   Edema of the scalp.
   Small hemorrhagic areas in the right cerebellar and right cerebral hemispheres.
   Hyperplasia of the spleen.
   Infarct of the spleen.

DR. MALLORY: From the anatomic findings it is very difficult to believe that there was not more variety in the symptoms which the patient presented. The chief finding was thrombosis of the right cavernous sinus extending backward through the longitudinal to both jugular sinuses down the right jugular vein. From the inferior end of that thrombosis an embolus split off, causing numerous pulmonary infarcts and also an infarct of the spleen. There were no other findings of any note. Nothing was discovered to suggest syphilis at all. The only culture made was from the spleen, unfortunately not from the thrombosis mass, and it showed no growth.

DR. CABOT: Is there nothing to show what made this thrombosis occur?

DR. MALLORY: I suspect that there must have been infection of the right orbit, although the orbit plate was not removed, so that we have no definite proof of that. The ethmoidal and frontal sinuses were negative, ruling out the other ordinary sources of cavernous sinus thrombosis.

DIAGNOSTIC FORUM

Based on the Cabot Case Histories selected and studied by the Yavapai County Medical Society and the Medical Officers of U.S. Veterans Hospital at Whipple, Arizona.

Any medical society or hospital staff in the southwest is invited to discuss these cases and submit their discussions or diagnostic conclusions for publication in this journal, along with those of the Yavapai County group.

CASES FOR YAVAPAI COUNTY MEETING OF DECEMBER 11th.

First Case:—Group 1.

An unmarried Irish-American woman sixty-seven years old entered the hospital March 17 in coma. The history is limited to a few facts obtained from a brother who did not live with her and from a nurse who brought her in.

For a year or more she had had attacks of dyspnea on exertion and her activity had been limited more or less. A week before admission she became rather suddenly ill with what seemed to be a respiratory infection. She had been in bed. The day before admission she seemed better. The day of admission she became very much worse. Halfway to the hospital she went into coma. There was no definite knowledge of medication except that her doctor gave her two pills containing digitalis early the evening of admission, and that before starting for the hospital she was given a quarter grain of morphia.

Clinical examination showed a woman obviously moribund lying propped up, comatose, breathing with great difficulty. Only a very cursory examination was done. Face and hands intensely cyanotic. Hands clammy. Profuse perspiration. Lungs examined only superficially over the front. In the upper right chest to the level of the fourth rib dullness, bronchovesicular breathing and many moist rales. Apex impulse of the heart not seen or felt. Left border of dullness 11 centimeters to the left of midsternum; fairly marked enlargement to the left. Rate rapid. Action regular. Sounds of fair quality, not muffled. At the apex a to-and-fro murmur, apparently a rub. Sounds at the base obscured by respiration. Pulses weak and thready. Artery walls not palpable. Blood pressure 100/60 to 180/100. Abdomen large, soft, tympanitic. Questionable liver edge palpable. (Nothing felt but a vague resistance.) Extremities cold. No edema. Pupils and knee-jerks normal.

No laboratory work was done.

Temperature 104.9°, rectal. Pulse 140. Respirations 40.

The patient was given caffein and adrenalin and propped up with her head high. Her color became better and her hands and feet warm. At ten o'clock in the evening she was given three grains of digifolin intramuscularly. This was repeated at 11 p.m. and at midnight. Her pulse slowed slightly and became stronger, and she seemed to recognize her brother. Caffein was repeated at 11 p.m. and 1 a.m. The blood pressure fell slightly and the pulse came down to 120 at one o'clock. A little over an hour later her respirations suddenly stopped. Her heart continued to beat for a few moments.

Second Case:—Group 3.

An American schoolgirl twelve years old was brought to the Emergency Ward October 22 in extreme prostration. The history was given by her parents, who were strikingly unable to answer any questions leading to a diagnosis.

Since October 17 she had not felt well, and on October 19 she stopped school. Her only complaints during the illness were lassitude, slight morning cough, loss of appetite and mild pain between the shoulders. She was thought to have been feverish for two or three days. October 21 it was noticed that her breathing was labored and rattling and that she was becoming cyonotic. That night the physician said her temperature was about 102°.

There was no history of operation, sinus, throat or dental symptoms, foreign body aspiration, marked headache, vomiting, mental disturbance, chills, sweats, hemoptysis, bleeding, severe pain or sore joints.

Physical examination was cursory because of her condition. It showed a cyanotic girl in semistupor, with prolonged expiratory rattle and irregular inspiratory gasps with clicking of the teeth. The throat was negative except for frothy exudate through which she breathed. There was no membrane. There was no tenderness over the sinuses. The chest was resonant throughout. The breath sounds were obscured by loud coarse moist rales of extreme pulmonary edema. Tactile fremitus and whispered and spoken voice were not elicited. The heart was negative except for a rate of 160 to 180. The pulse at first was of fair quality, better than her appearance indicated. The abdomen and extremities were negative except for cyanosis.

The temperature and respiratory rate are not recorded.

She was put upon the dangerous list immediately, and was given atropin, nitroglycerin, adrenalin, caffein and digitalis. Venesection was attempted. In less than three hours she died.

PERSONALS AND NEWS

DR. JOHN W. STACY, who has been resident physician at the Good Samaritan Hospital in Phoenix, has associated himself with Drs. Ketcherside and Lount of Yuma, with offices in the Yuma National Bank Building.

THE SOUTHWESTERN DENTAL SOCIETY held their annual convention in Tucson October 28 to 31, with a very large and enthusiastic attendance. Their four days of scientific program was enlivened by many social events.

ST. MARY'S HOSPITAL of Tucson has been given a Class A rating by the American College of Surgeons. This is probably the oldest hospital in Arizona, having been established forty-eight years ago.

DR. MORTON J. KIMBUL, of Phoenix, after spending the summer in the northwest, has returned to Phoenix and reopened his offices in the Security building.

DR. MAYO ROBB, of Phoenix, who took charge of the practice of Dr. D. F. Harbridge during the summer months, has located permanelty in Phoenix, being associated with Dr. H. T. Bailey.

DR. GEORGE SHIELDS, of Yuma, has removed to Phoenix and will be associated in practice with Dr. Charles S. Vivian, their practice being confined to urology and surgery.

DR' ROSS MOORE, Wilshire Medical Building, Los Angeles(after January first), has arranged to make available to the medical profession the strain of tertian malaria which he has been using for two years, in the treatment of late syphilis. Those interested in such treatment can secure further data from Dr. Moore at 520 W. Seventh St., Los Angeles, up to the first of January.

Southwestern Medicine

Printed by THE A C TAYLOR PRINTING CO, Phoenix, Arizona
Published monthly for the Board of Managers of the four constituent societies.

Volume XII. NOVEMBER, 1928 No. 11

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AN UNUSUAL OPPORTUNITY FOR MEDICAL SOCIETIES

One of the great problems of small county medical societies is to secure something interesting for their meetings. The few members are usually busy, they know each other's opinions fairly well, and unless something from outside of their circle comes in to weld them into a unit, they will seldom cohere.

In presenting the idea of an open forum in diagnosis, with interesting cases selected by a group of doctors in Yavapai County, it is hoped that we may stimulate interest in clinical study and clinical discussions. A county medical society of five members or twenty-five members could find opportunity twice a month or once a month to meet for two hours, and discuss clinical problems as presented by actual cases. The advantage of using the cases published in this journal will be that we may compare notes, and that we may learn what others think about the same cases which we have studied.

The Massachusetts General Hospital is taking considerable interest in our attempt to popularize clinical case discussions in Arizona and the southwest. They now offer to send to any hospital staff or medical society records of cases that have not yet been published, together with the necropsy findings. If any society or group in the southwest are interested in such a proposal as this, please write to this journal and arrangements will be made.

AUTOPSY FINDINGS IN DR. J. I. BUTLER

(It was the dying request of Dr. J. I. Butler, of Tucson, Ariz., that a thorough post-mortem examination be made of his body and the results given to the medical profession for whatever value it might have to them.

Dr. Butler first developed symptoms in the left shoulder area about February, 1928. This was in the form of a soreness which inhibited certain movements of the shoulder. After this had persisted for two or three weeks, he made roentgenograms, which showed definite changes in the bone of a destructive type. Consultation with Dr. Ellis Jones, of Los Angeles, developed the almost certain diagnosis of a malignant lesion in the upper humerus. Soon after this the axillary and supraclavicular glands became enlarged and tender. One of these removed for diagnosis showed the structure of sarcoma.

Roentgen and radium therapy, with injections of colloidal lead, failed to check the progress of the disease and Dr. Butler succumbed during the last of June. The following is detailed report of the autopsy findings made by Drs. Brem. Zeiler & Hammack, of Los Angeles.)

Middle aged white male, fairly well developed and nourished. Slight subcutaneous edema of right leg and ankle. In the left supraclavicular space there is a lobulated soft mass and the skin over it presents a surgical scar. There is also an old suprapubic scar. No other subcutaneous lymph nodes palpable.

In the left axillary space there is a large lobulatde non-infiltrative soft tumor mass, the cut surface of which is homogeneous and grey white. Encircling the upper third of the left humerus is a tumor mass about 2 cm. thick. The periosteum is completely destroyed. The cortex has a moth-eaten appearance and on the medial surface just below the neck there is an eroded area of the cortex extending into the medullary cavity. The lower portion of the joint capsule is destroyed but the articular surfaces are not involved. Practically the whole of the medullary cavity is filled with soft tumor tissue, the head is almost completely filled with tumor, there being a few bony trabeculae extending through it. There is a pathological fracture at the lower end of the upper third of the humerus.

THORACIC CAVITY

In the superior and anterior mediastinum are several large soft tumor masses averaging about 5 cm. in diameter. In the hilar regions of both lungs

are similar masses, those on the left being larger. Extending along the secondary bronchi there is a sheath like infiltration. Those masses on section are soft and have a homogeneous grey white appearance; the capsules are intact. Sections through the lungs reveal a few scattered small circumscribed masses especially in the left lower lobe. Each cavity contains about 500 cc. of clear fluid. The pericardial sac is slightly enlarged and there is a moderate increase in fluid. In the epicardium on the anterior surface of the right ventricle is a mass about 1 cm. in diameter; the whole of the base of the right auricular appendage is filled with a soft mass. No valvular lesions demonstrable. The heart muscle is pale and flabby.

ABDOMINAL CAVITY

It contains about two liters of pale milky fluid; the liver edge is 6 cm. below the costal margin. The spleen is irregularly shaped and enlarged. There is a large retroperitoneal mass in the left kidney region. Surrounding the celiac axis is an irregularly lobulated mass about 15 cm. in diameter; extending along the splenic artery is an elongated mass and one also in the hilus of the spleen. All the pre-aortic lymph nodes are greatly enlarged and completely filled with tumor. In the left wall of the pelvis there is an irregular lobulated mass behind the peritoneum.

Liver: Moderately enlarged; just beneath the capsule are a few scattered, slightly raised cirdumscribed tumor nodules averaging .5 cm. in diameter. On section a few similar nodules are seen scattered throughout the parenchyma. Gall bladder and ducts are negative.

Spleen: Is irregularly shaped, the lower pole being enlarged and globular shaped and within it is a globular shaped mass about 8 cm. in diameter. On section this mass is hemorrhagic and friable. In the upper portion there are several smaller masses.

Pancreas: No tumors within it but extending along the splenic artery are several enlarged lymph nodes.

Left kidney: Completely surrounding it is a mass about 2 cm. thick. The surface of this is smooth and the mass is apparently encapsulated. On section the mass is fairly soft and compact; there is no invasion of the kidney. The tumor surrounds the left ureter in a sheath-like manner throughout its entire extent, the ureter averaging 1.5 cm. in diameter. The wall is thickened and the lumen is small but patent. Sections reveal no masses within the kidney but tumor infiltration extends along the ramifications of the pelvis.

Right kidney: Moderately enlarged, several nodules 1 cm. in diameter beneath the capsule. Section shows several nodules throughout the parenchyma. Right ureter negative.

Urinary bladder: Within the left wall in the region of the ureter is a tumor mass about 1 cm. thick. but this has no connection with the prostate.

Prostate: The middle lobe has been removed, the lateral lobes are hypertrophied but no tumor mass is within it. Just posterior and to the left of the prostate is a mass about 5 cm. in diameter. This is just anterior to the rectum but does not extend into it. All of the deep iliac chain of lymph nodes are greatly enlarged and on section are soft and filled with grey tumor.

Stomach, small and large intestine were negative, Adrenals are negative.

ANATOMICAL SUMMARY

Large, soft and lobulated tumor mass in left axilla.

Tumor surrounding upper third of left humerus.

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a—Erosion of cortex on medial surface of humerus.
b—Narrow cavity filled with tumor.
c—Pathological fracture in lower portion of upper third. Subcutaneous tumor mass in left supra-clavicular space. Large soft tumor mass in superior and anterior mediastina. Numerous tumor nodules around celiac axis and in pre-aortic region.

Two tumor nodules in epicardium of heart. Tumor noduies in spleen, liver and kidneys. Large mass surrounding left kidney and ureter. Tumor nodules in lungs.

MICROSCOPIC

Sections were made from various portions and they all have essentially the same appearance. The tumor is a cellular one with a diffuse growth of small lymphoid cells lying in a fine reticulum. The reticulum is irregularly distributed, being absent in some portions, but in others it is fibrous. Numerous blood vessels extend through the reticulum but these appear to play only a passive part. There are numerous scattered larger round cells of the reticulum type, also multinucleated cells resembling Dorothy Reed cells as well as a few cosinophiles and plasma cells. A rather peculiar feature is the almost complete absence of mitotic figures.

In the spleen and other organs the tumor is sharply circumscribed and is encapsulated.

We feel that this tumor is probably of lymphoid origin and are inclined to consider it as a lymphsarcoma.

EL PASO COUNTY MEDICAL SOCIETY
(Sept. 24, 1928)

The El Paso County Medical Society met September 24, 1928, with twenty-one members and three visitors present.

DR. E. J. CUMMINS reported a case of dislocation of the ankle bones resulting from a fall on the knee. The tendon of the tibialis anticus was displaced between the internal and middle cuneiform homes. X-rays of the ankle were shown.

DR. RALPH HOMAN presented a paper on "Pulmonary Emphysema" with a case report. The case was formerly diagnosed bilateral spontaneous pneumothorax, and the x-ray pictures supported this diagnosis. The symptoms began after strenuous rowing. The patient died, and autopsy showed extremely large empysematous sacs on both upper lobes. The heart showed coronary thrombosis, which is thought to have caused death. Dr. Geo. Turner presented the autopsy material, and the case was discussed generally. Dr. Awe stated that he had reported the case to the pathologist at the University of Iowa, who told him that out of 3,000 autopsies performed by him there had been no such extensive case of emphysema, and that there was undoubtedly a marked hereditary weakness of the tissue as well as violent exercise have produced such a condition.

DR. HILL reported on the testing of cows in El Paso county for tuberculosis. Out of more than 9,000 tests there were seven positive reactions. Those cows were slaughtered.

DR. BRANCH spoke of the successful campaign in Dona Ana county, New Mexico, against tuberculosis cattle.

DR. LAWS suggested that if Drs. Hill, Branch and Outlaw had any plan by which the society could aid in this work, they bring their suggestions before the society.

DR. MILLER moved that Drs. Branch, McCamant, Outlaw, and Hill be appointed to bring such suggestions and recommendations before the society. This motion was seconded and carried.

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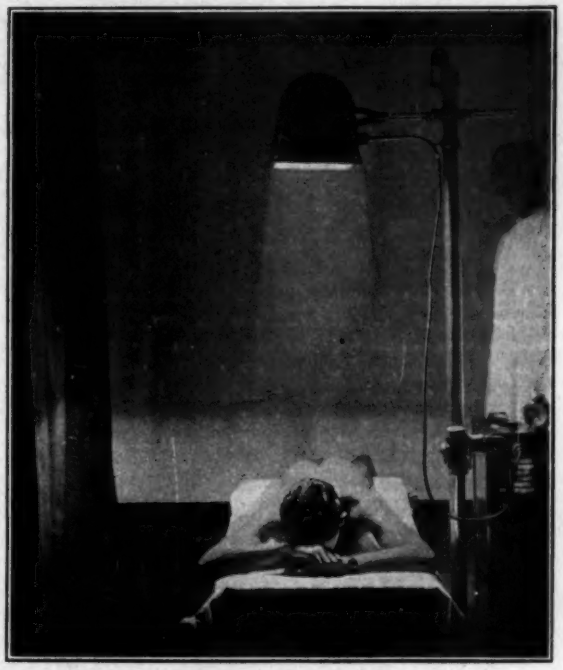
—E. P. CUMBERBATCH
M.B., D.M.R.E., M.R.C.P.

From a paper read before the Southport Division of the British Medical Assn., March 30, 1928. (British Med. Jour., July 14, 1928)



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DR. AWE spoke of the work among tuberculous cattle in Iowa.

The secretary presented a letter of transfer of Dr. S. D. Armistead from the Baldwin County, Alabama, Medical Society. This was referred to the committee on credentials.

(October 1, 1928)

The El Paso County Medical Society met October 1, 1928.

DR. F. C. GOODWIN presented a paper on "The Weak, Painful Foot." A resume of the paper follows:

1. The anatomy of the foot was reviewed with illustrations to show the longitudinal and transverse arches of the foot. The normal ranges of motion were demonstrated in the foot and ankle, also the joints in which these motions were produced.
2. The functions of the feet were explained in detail. These are two: They serve as a passive support and as a propeller. To illustrate this, various models and the bony foot were shown.
3. After considering the foot as above stated it was taken up as a complex mechanism. To function properly as a mechanism the bony structures must be held in physiological alignment by the ligaments.
4. Order of examination was outlined and reasons why such an examination should be systematic. The patient is examined with, and without shoes. The shoe is studied to see the areas which are being worn.
5. Treatment consists of straps and pads for four weeks. These are changed once weekly. Plaster of Paris models are made from the feet. To these the Lowman or the Whitman arch support is constructed.
6. Three cases were reported showing results.

(Oct. 15, 1928).

There was a regular meeting of the El Paso County Medical Society, October 15, 1928. A symposium of venereal diseases was given.

The first paper: "Prevalence of Venereal Diseases Before and Since the War," by Dr. K. D. LYNCH. Dr. Lynch gave an informal discussion on this subject. He stated that a review of voluminous statistics failed to convince him of any special improvement. Immediately after the close of the war certainly something had been gained by education of the troops of the A.E.F. Apparently a new crop of young people no longer cognizant of venereal disease dangers were supplying the bulk of the patients. The percentage of his own venereal disease cases at present represented an exact duplicate of the pre-war conditions. However, he believes that syphilis is not so prevalent locally. The regulations for reporting venereal diseases he believes to be ineffective as a deterring agent.

The second paper: "The Control of Syphilis and the Prevention of After-effects," by DR. LESLIE M. SMITH. The question of whether syphilis is increasing is a difficult one to answer. Unquestionably syphilis has received more attention since the war, due to the greater experience with this disease obtained by physicians who served in the army and navy, and to the education of the soldiers and sailors concerning the venereal diseases. The routine Wassermann test which is now performed in many clinics and hospitals has aided in detecting many cases of latent syphilis which

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would otherwise have gone unsuspected. Whether there is an actual increase in the number of infections year by year or not, certainly more cases are being discovered and treated. During the present century the incidence of a number of infectious diseases has been decreased, and some have been almost abolished in localities where they formerly were prevalent. To predict that syphilis might likewise be abolished one might appear over-optimistic. However, I believe the task is not impossible, and it should be the goal for which we strive. There are several factors which must play a part in the control of this disease and in its eventual abolition.

First in importance is education,—this to include medical students, physicians, nurses, parents, boys and girls. The instruction on syphilis has been entirely too meagre in mot medical schools, with the result that many of our present practitioners were inadequately trained for the proper management of syphilis. This is gradually being overcome by the medical schools themselves, and by the short postgraduate courses and clinical conferences being held every year under the auspices of various organizations. While nurses need not be syphilologists, much can be accomplished by instructing them in public health aspects of syphilis. Instruction can further be given in talks by physicians before parent-teacher assemblies, and by physicians and nurses before separate groups of the sexes in school. These talks to parents and to boys and girls should be given by those who are especially equipped with teaching ability and tact, for the laity are accustomed to thinking of sex and venereal matters as subjects only to be whispered. The selection of a lecturer who has not this ability would be fatal to the purpose in view.

The instruction in venereal prophylaxis given the men in the service during the war was undoubtedly a good thing; and if there has been an increase in syphilis it has been in spite of that instruction. Venereal prophylactic methods might well be given more publicity.

The various state laws requiring reporting of syphilis and other venereal diseases are a step in the right direction, but so far they are not supported and are probably of very little benefit. If the law is explained to all patients with syphilis, the knowledge that they may be reported for failure to take treatment may perhaps have its influence.

The economic importance of the late and disabling phases of syphilis is worthy of consideration. A large percentage of the paralytic and insane objects of county and state charity owe their condition to syphilis, and usually mistreated or insufficiently treated syphilis. Most of this trouble can be prevented by the thorough treatment of early syphilis, follow-up of the cases, and rigid tests of cure before a final discharge. Those tests should include repeated blood Wassermann tests, physical examinations, spinal puncture, and complete examination of the fluid. It is up to us as physicians to give instruction to all our syphilitic patients, regarding contagiousness, the amount of treatment probably necessary, the prognosis of the disease at the stage at which the patient happens to be, and the necessity for rigid tests and observation afterwards.

The third paper, "The Effect of Gonorrhoea on Parturition" by DR. HARRY LEIGH.

The effect of gonorrhea on parturition is essentially that of a classical gonorrheal infection plus certain grave modifications due to the increased vascularity of the pelvic tissues. The possible effect of gonorrhea on the parturient must be viewed from several angles.

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First, what is the effect on the incidence of pregnancy and the occurrence of sterility? Modern sterility studies place the blame most frequently on the male. Gonorrheal infection ranks first as the cause of sterility in the male. At the same time that an obliterative vesiculitis has rendered the male sterile, a diseased prostate may in turn infect the wife and subsequently render her sterile. Lesions in the female may be absolutely incompatible with impregnation, such as is found with tubal obliteration and systic degeneration of the ovaries. Lower down obliterative cervicitis or a chronic endocervicitis, or an endometritis may render implantation or the welfare of the fertilized ovum impossible. Again excessive purulent discharge may produce a condition incompatible with the life of the spermatozoa.

Next gestation is often stormy in the presence of a gonococcic infection. In the primiparous, an initial gonococcic infection with a simultaneus pregnancy is apt to be a serious affair. Gynecological invalidism is almost certain. Ordinarily in practically all cases the gonorrhea elects the vulvovaginal glands, cervix and Skene's ducts. Due to the added succulence of the tissues the epithelium may be severely attacked; green-yellow pus exudes, the mucosa becomes reddened and ulcerated, and frequently condolomata develope, the vaginal membranes bleed to touch, the cervix is hypermic, pouching and eroded; foul muco-purulent material exudes; direct extension frequently infects the decidua. Such cases may abort. Further extension may involve the adnexa. A septic course frequently supervenes.

Labor itself may be associated with dystocia resulting from an old pelvic cellulitis, a perimetritis, or an endometritis. Trauma in the vulvovaginal canal is certainly apt to be more marked in the primipara, due to the thickened inflamed tissues. Such birth injuries are slow to heal. The old-time thirteen day temperature is still found in the indolent healing of a gonococcic infected vaginal canal. Direct extention is especially prone to occur.

The puerperium is occasionally associated with sub-involution. Obliterative peritonitis, salpingitis and a pan-pelvic cellulis may occur. However we are frequently astounded at the mildness of post-partum reactions in the presence of extensive gonococcic infections. However gynecologic invalidism is very common.

Of practical importance is the resistance to treatment found in the average parturient. Extreme difficulty is always experienced in securing a negative slide or a clinical cure.

Important are possible sequellae that may occur as complications. Joint involvements of persistent character, pyelitis and endocarditis are not uncommon. Accidental eye infections of the patient or of contacts are frequent. Gonococcic proctitis from enema or douch tips can easily disseminate the disease. Due to excessive vaginal secretions tenement toilets frequently convey the infections to young girls. Many such cases have come to my attention. Hospital epidemics of gonorrhea often start from an infected obstetrical patient.

The eyes of all new-born should get special attention wherever a suspect has been delivered, but especially those of a gonorrheal mother. The practice of adopting less irritant drugs, I believe, is to be condemned. One to two per cent silver nitrate has proven effective and safe. Opthmologists estimate that 20 per cent to 30 per cent of all blindness is due to gonorrhea in the new-born. In our American institutions for the blind today, something like 25 per cent of all blindness is due to gonococcic infection.

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sents certainly public health and personal welfare problems.

1. A sterility problem extremely difficult to solve.
2. A sterility problem particularly apt to result in domestic crises.
3. The problem of the gynecologic invalid and her concomitant economic dependability.
4. The abortion problem.
5. The late temperatures of puerperium.
6. Communicability of gonorrhea where children are present.
7. Hospital epidemics of gonorrhea.
8. The public liability for the gonococcic blindness.

The fourth paper, "Prostitutes and Prostitution," by DR. E. D. STRONG.

Dr. Strong said in discussing this subject that a brief review of the historical background and a comparison with the present can only lead to one conclusion, namely, that prostitution and the prostitute is here and has always been. Marital incompatibility and perversion of the sex instincts or requirements maintain the ever-present practice of prostitution. He maintains the point that both male and female prostitutes must be recognized as such. Married men as well as married women constitute a very large per cent of offenders, particularly the former.

The fifth paper, "The Suggested Method of Control," DR. W. R. JAMISON.

The history of prostitution can be traced back to the earliest traditions of the human race. Moses attempted to eradicate prostitution without success. Among the Greeks and Romans this condition was rife. The history of prostitution can be traced down the ages. As surely as a community of any size is gathered in a given locality, just as surely will the prostitute make her appearance. The number of prostitutes will bear a definite ratio to the number of able bodied unmarried men in any given community.

It is impossible to promulgate laws that will control sexual appetite,—one of the strongest, if not the strongest, instinct of the human race. As a result of many economic and social factors, 'there has arisen in society a figure which is certainly the most mournful, and in some respects, the most awful, upon which the eye of the moralist can dwell. That unhappy being whose very name it is a shame to speak; who counterfeits, with a cold heart, the transports of affection, and submits herself as the passive instrument of lust; who is scorned and insulted by the vilest of her sex, and doomed, for the most part, to disease and abject wretchedness and early death, appears in every age as the perpetual symbol of the degradation and sinfulness of man. Herself the supreme type of vice, she is eventually the most efficient guardian of virtue. But for her, the unchallenged purity of countless happy homes would be polluted, and not a few who, in the pride of their untempted chastity, think of her with an indignant shudder, would have known the agony of remorse and despair. On that one degraded and ignorant form are concentrated the passions that have filled the world with shame. She remains, while creeds and civilization rise and fall, the eternal priestess of humanity, blasted for the sins of the people." (Lecky. History of European Morals.)

In the statement above is contained the nucleus of the whole subject of control of prostitution. It is impossible to regulate the sexual instinct. Only by education can we seek to prevent venereal diseases, and that education should begin with the young boy or girl. Instruction in sexual matters should be given early in life, not bluntly, but by

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drawing the child's attention to the pollenization of plants, the hatching of eggs, etc., and gradually, as he or she becomes older, bringing out the propagation of the species, animal and human. When it is judged that the young girl or young man has arrived at an age when they can understand it, the subject of venereal diseases should be taken up, the dangers pointed out and the manner of contracting the same explained. The proper teachers of this are the parents. Many parents are horror stricken at the idea of explaining these things to their progeny, in which case the services of the family physician may be asked. The gradual instruction of the child in sexual matters brings him or her to manhood or womanhood with a commonsense attitude towards these conditions, and they avoid learning them through salacious conversations at school.

What are the underlying causes of prostitution? First, lack of education in sexual matters. Second, inability to live on the wages paid to women. Third, love of finery.

The first cause has been discussed in the preceding paragraph. The remedy for the second is self-evident, and there is no means of preventing a girl selling her body for money to adorn it.

It is Utopian to dream that prostitution can be abolished. From the beginning of history various rulers have struggled with this problem, even to the extent of providing a death penalty for any woman who would prostitute herself. It must be regarded as an amni-present evil. Rules and regulations have been promulgated governing prostitutes in various countries, but these have failed to stop the traffic in human bodies. Within the last ten or fifteen years in this country, we have officially refused to recognize licensed or regulated prostitution, but it has not solved the problem. Loose women have dispersed all over this city, they ply their trade in hotels, rooming houses and in the so-called respectable residential sections of the city. The advent of the motor car provided another means for the professional prostitute to exploit her charms, but without the advantage of having sanitary appliances at hand to prevent infection from her customer.

Another and unexpected effect of taking off the regulations of prostitution has been the seduction of young girls by boys of their own age, due to the fact that the young man has no place that he can go to satisfy his sexual urge. Ten or twelve years ago, every young man knew where the red-light district was and its purpose. Nowadays, a motor car, a flask and a pretty girl and the deed is done. Another girl has been ruined and may, by force of circumstances, be forced into the ranks of prostitution.

Harwood, (J.A.M.A. Dec. 22, 1906), tells of regulation in a settlement of steel workers, the force of which was crippled by venereal disease. Medical examination and cooperation with the keepers of brothels produced good results. Houses were licensed, the inmates rigorously examined by competent physicians. This plan requires the consent of the American people to the legalization of prostitution, which I am afraid would not be tolerated. The strife, as it at present exists, between the abolitionist and the regulationist, is a fruitless battle. Arguments as to the advisability of devising some means for lessening venereal disease may easily demonstrate conclusions on one side, but sentiment and conventionality are equally powerful in formulating contrary conclusions.

Results in large cities have not been what they should have been, largely due to the fact of political interference and graft. But in smaller communities regulation seems to have worked out fairly well. This is best seen in the army.

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To prevent venereal disease, prophylaxis stations should be established in several part of the city where young men can take treatment immediately after intercoure. Of course there will be a loud protest from the ultra-good, that this puts a premium on immorality. What of this? Is not a clean immoral young man better than one who has lost both health and morals? The advantage of prophylaxis stations is seen in the results in the army.

When the great American people can tackle the problem of prostitution and its results, venereal disease; look the matter squarely in the face without bigotry, without fanaticism and recognize the fact that this condition is with us to stay, then a great forward step wil have been taken toward the diminution of venereal disease.

Let the abolitionist and the regulationist ge. together on the subject. Let them look the facts in the face. Then with the greatest tolerance for each other's opinions and beliefs, formulate a plan that will in some measure control prostitution, and, in so doing, abate the awful menace of venereal disease.

DISCUSSION

DR. OUTLAW, City Health officer, spoke of the recent clinic report where forty new cases were reported, all but three were for syphilitic infection. Reporting is the law and facilitates check in refractory patients who are a community menace. The cases are not reported by name. Police enforcement is to be enlisted to bring such persons to treatment if necessary.

DRS. JAMISON, SHANNON, SWOPE, STRONG, WERLEY and LYNCH all expressed doubt as to the value of reporting disease. Education and prophylactic stations were urged with proper publicity. Proper education, they all agreed, offered the best solution. Dr. Strong spoke to considerable length on the technique of prophylaxis versus postphylaxis.

(Oct. 29, 1928)

The El Paso County Medical Society met October 29, 1928.

DR. H. D. BARNARD, of Los Angeles, discussed informally fractures of the end of the humerus.

Prefacing his remarks, he stated the average run of these fractures show unsatisfactory end results. For three years he has been engaged in standardizing the management of these cases at his Los Angeles hospital.

Prognosis can best be determined in terms of functional recovery, and economic capacity of the injured after a year. The anatomic result may be of less moment. Children, as a rule, recover better than adults. A 90 per cent arc of movement and function may be classed as a 100 per cent perfect. The time element in reduction after the initial injury may greatly influence the prognosis. The T type injury should be accompanied by a warning of a possible poor function. External condylar fractures may result in ulnar nerve dysfunction from scarring or direct injury.

Treatment must be in accordance with the type and age and the severity of the injury. Mechanically the block and tackle should be used to replace fractures more than two hours old, and then under ether anaesthesia. With older fractures, if greatly swollen, and if associated with tropic blisters, a period of suspension to permit the joint and soft tissues to improve is imperative for a

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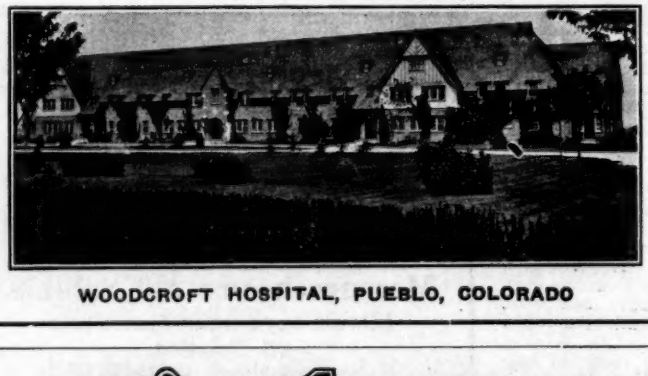

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good deposition. In reduction, an increase, first, of thed eformity, then flexion, and external retation so the little finger is on a line with the external deltoid fibers, is correct to prevent rotation of the condyle.

Dr. Barnard employs a moulded plaster splint to maintain the arm. This splint is made of sixteen layers of bandage and extends from the opposite illiac crest to the shoulder of the affected side. It then extends down to the external side of the humerus, over the olecranon to the palmar surface of the hand. Bandages are applied in the reverse order, care taken to protect the radial pulse.

Dr. Barnard showed diagrams of the use of Coen's line in determining a prognosis.

DR. W. L. BROWN spoke on the usefulness of the method and substantiated the preference of a closed reduction.

DR. GOODWIN recited a case of a biceps hematoma which he had recently seen.

PERSONAL NOTES

Drs. R. B. Homan, Miller, and Safford attended the district medical meeting at Sweetwater early in October.

Dr. Snow of Abilene spent some time in El Paso late in October.

Dr. G. Werley recently paid Dr. McKinley of Alamogordo a professional visit. Dr. McKinley has been ill for several months.

Dr. Hugh Crouse continues confined to his residence and unable to attend to any professional duties.

A recent visit of the secretary of the **Medical Milk Commission** here resulted in a complete reorganization ofo ur previous method of handling certified milk committee appointments.

JOURNALS FOR SALE:—Dr. Elliott C. Prentiss of El Paso offers for sale some journals which should be of value to some of our readers. He has the American Journal of Tropical Medicine complete from Vol. 1 to date which he will sell. He has the Washington Medical Annals complete from Vol. 1 up to the time it was discontinued. He has the New Mexico Medical Journal complete except for about half of Vol. 5 and two numbers since then. Anyone interested may write to him at 515 Roberts-Banner Bldg., El Paso.

The Department of Health report the following deaths and births:

Deaths for Month of September, 1928

	Male	Female	Total
White	31	15	46
Mexican	46	41	87
Black	2	1	3
Totals	79	57	136

Births for Month of September, 1928

	Male	Female	Total
White	43	28	71
Mexican	84	88	172
Black	1	2	3
Others	2	0	2
Totals	130	118	248

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